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The Medico-Legal Applications of Blood Grouping Tests

The issue of the *Tentative Supplementary Report on the Medicolegal Applications of Blood Grouping Tests*[1] is likely to raise acutely the controversial matter of the most appropriate Rh blood group system nomenclature.

In a recent Editorial[2] we discussed this problem and drew attention to the powerful case which had been made out for Wiener's international terminology. Since the forensic significance of the blood groups depends essentially on a synthesis of genetic and serological facts, the question of nomenclature is not merely one of personal preference. On a scientific basis the case for the recognition of Wiener's terminology to the exclusion of all other notations is overwhelming. The authors of the CDE nomenclature with its corollary of a one-to-one correspondence between gene, antigen and antibody have themselves been led to abandon the props which they invented to sustain their theory, e.g. 'gene deletions' as in -D-; and the variable anthropological distribution of the CDE and CdE chromosomes as evidence of crossing-over. Even such ardent supporters of the Fisher-Race theory as Dunsford and Bowlly[3] have recognized the fundamental fallacy in this concept. These authors state: 'For normal blood group work this one-to-one theory is both tenable and practicable, but research has shown it to be not strictly true.' The medico-legal implications of such a facile departure from strict scientific discipline are too obvious to require further emphasis.

The publication of the *Tentative Report* provides an opportunity for suggesting an extension of its scope beyond purely technical matters and the inclusion also of the following features:

Emphasis on the necessity for the use of potent serological reagents (the specificity of which should preferably have been confirmed independently by some recognized authority) and the use of positive and negative controls in testing.

Mention of the use of high titred group O serum in ABO testing, since weak sub-groups of A (A_3 and A_4) may otherwise escape detection. These weak sub-groups of A are not infrequent in Negroes and may be accompanied by specific anti-A isoagglutinins, albeit of low thermal amplitude.

The *Report* suggests that the results obtained with univalent antisera (i.e.

1. Published elsewhere in this issue by permission of the Committee on Medicolegal Problems of the American Medical Association and reprinted (with acknowledgments) from the *Journal of the American Medical Association* of 19 May 1956.
2. This Journal (1952): **2**, 193.
3. Dunsford and Bowlly (1955): *Techniques in Blood Grouping*. Edinburgh: Oliver and Boyd.

sera which do not react with cells suspended in saline and which require techniques such as the antiglobulin test, the conglutination method or the use of enzyme treated cells) are of little use in medico-legal blood tests. This limits the application of blood grouping tests severely. The limitation is unjustified if potent and specific reagents are used.

With the exception of the Lewis blood group system, all blood factors so far discovered express themselves in a Mendelian fashion as dominants (or as co-dominants in the case of reciprocally related factors). Thus, in a case of disputed paternity, the presence of a blood factor (S, Fy^a, He, etc.) in the child's blood excludes paternity if it is absent from the bloods of the mother and of the putative father. This is an inescapable application of Bernstein's original principle. The fact that the precise relationship of the particular blood factor to other factors in the same blood group system may not yet have been worked out in detail, does not detract from the validity of this simple principle in the exclusion of paternity.

Ethical as well as legal issues often confront the practitioner who performs blood group tests and a short section on these aspects of the work of the blood grouping expert would considerably enhance the value of what will undoubtedly become a standard reference work on the subject. Important problems arise in a case such as the following:

A man is found to have complete aspermia. He divorced his wife two years before and (though he has remarried) he is supporting a child by the first wife. He was always under the impression that it was his own child; but as a result of the laboratory investigation of his semen, he now suspects that it is not.

The divorce order gave the mother the custody of the child, but he is allowed to visit at certain intervals. He plans to take the child for a blood test without the mother's knowledge and to have his own blood examined.

The chances of exclusion in these circumstances are, of course, fairly slender but he and his second wife want the tests done because of the now complicated question of continuing to pay maintenance for the child.

Is an examiner ethically entitled, however, to undertake a paternity test without the full knowledge and consent of both the alleged parents (regardless of a Court order—since this is binding on the disputants only and not on the examiner)? Is it not ethically preferable for the examiner to do the tests on the understanding that he will furnish identical reports impartially to both sides? Is it ethically proper for a practitioner to examine the blood of a child without the knowledge and consent of one of its alleged parents who may have the legal custody of the child?

What complications arise from a differentiation between custodianship and guardianship of the child in dispute?

This *Tentative Report* is a superb review of present-day knowledge of blood groups in relation to medico-legal issues. It can be anticipated that the only criticism it will receive will come from quarters hostile to the adoption of Wiener's Rh nomenclature; but it would be a great pity if terminological views (adhered to with passion rather than reason and which do not reflect the established body of scientific knowledge) should be allowed to disturb the international adoption of this valuable *Report*.

The A.M.A. Committee on Medicolegal Problems would welcome comments from workers in the field, so that the preparation of a final report can be considered by the end of the year. Communications should be forwarded to the Executive Secretary of the Committee on Medicolegal Problems, 535 N. Dearborn St., Chicago 10, I11.

Suicidal Laryngectomy

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Suicidal injuries, especially throat wounds, vary greatly in their nature and severity. This has always been the experience of forensic pathologists. The case here presented shows to what lengths a suicide will go to attain his purpose.

Case Report

The victim was a potter's polisher, 50 years of age. Two years before he had been medically examined and found to have a mild degree of silicosis. Thereafter he became depressed and moody but continued with his work until a few days before he died. When on the morning of 19 November 1954 his wife left the house to go to work he was still in bed and 'in his usual health'. When she returned home at 3 p.m. the house was locked and bolted from inside. She then went to fetch a relative who lived some distance away and came back with her to find the house still locked. Finally a policeman was summoned who broke into the house at 3.45 p.m. He found the man lying dead on the floor of his bedroom with a large wound in his throat. Close to him lay a blood-stained saw-edged bread knife and what he described as 'a portion of the inside of his throat'.

Necropsy was performed at noon on the following day. The cause of death was obviously a large incised wound in the front of the neck measuring 10 cm. from side to side with edges which were jagged and slightly bruised. At its left extremity there were 5 tentative incisions running parallel to the main wound, some above, others below it (Fig. 1). The cut extended through the skin and subcutaneous tissues, the sternohyoid and sternothyroid muscles and the trachea, exposing the anterior wall of the pharynx and the oesophagus. The wound thus appeared as a large wedge-shaped gap in the neck whose edges were 8 cm. apart when the head was allowed to fall backwards. The roof of this gap was formed by the floor of the mouth, the epiglottis and the hyoid bone. Below it was the cut

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Fig. 1. Throat wound and larynx as found by the police.

upper end of the trachea, which had retracted somewhat, and most of the thyroid gland.

The whole of the larynx and the upper pole of the left lateral lobe of the thyroid were missing, but they were subsequently produced by the police constable who had discovered the body (Fig. 1).

The common carotid arteries and internal jugular veins had been spared; only the right external jugular vein was cut. Both sternomastoid muscles had been incised but not completely severed. There were no other injuries.

Internal examination showed fairly extensive nodular silicosis throughout both lungs with bullous emphysema at the apices. The hilar lymph nodes contained both tuberculous and silicotic lesions. The heart was normal in size. There was no other relevant pathology.

Discussion

Evidence given at the inquest leaves little doubt that this was a case of suicide. The appearance of the wound with its tentative cuts at the left extremity is consistent with this assumption. The ferocity with which some suicides carry out multilating injuries is too well known to require further emphasis. Professor Sydney Smith's case,[1] who inflicted 7 separate wounds upon himself, and the case described by Kerr[2] where a man completely severed the transverse process of a cervical vertebra, are but 2 examples. Littlejohn (quoted by Smith[3]) reported the case of a woman who had torn out the right lobe of her thyroid gland.

I have found no reference, however, of a complete excision of the larynx as in the present case. Comparison with another larynx which was removed in a more orthodox fashion by an otologist (Fig. 2) shows the

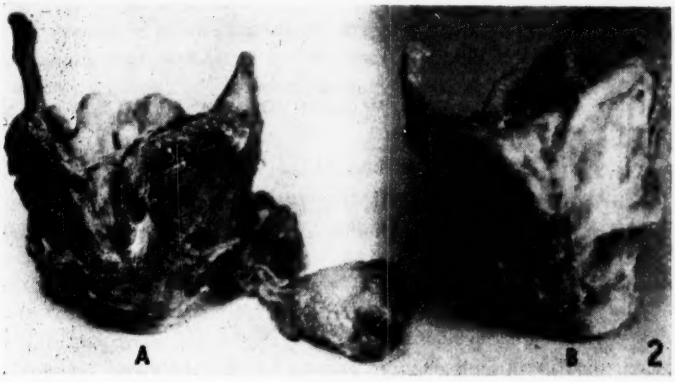

Fig. 2. Larynx removed by suicide (A) alongside a surgical laryngectomy specimen (B).

astonishing precision with which this suicidal laryngectomy was performed. Closer examination suggests that it was achieved with but 2 sweeps of the bread knife found beside the body after death. The anterior cervical muscle had been dissected away from the thyroid cartilage and the superior cornua remained almost intact. It is probable that death was the result of haemorrhage and traumatic shock, though it is hard to say how long the victim survived his mutilating operation.

Summary

A case is described of a suicide who, with a bread knife, cut his throat and excised his own larynx.

My thanks are due to Mr. G. W. Huntbach, H.M. Coroner for Stoke-on-Trent, for permission to publish this case and to Mr. C. H. Lees for the photographs.

References

1. Smith, S. (1926): Lancet, **2,** 1163.
2. Kerr, D. J. A. (1924): Brit. Med. J., **2,** 1042.
3. Smith, S. (1945): *Forensic Medicine*, p. 157, 8th ed. London: J. and A. Churchill, Ltd.

An Unusual Wound of the Heart

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Medical literature abounds with accounts of unusual rectal mishaps and injuries. The oddest foreign bodies may gain access to the rectum and cause penetrating wounds. The case to be described is unusual in that an 'instrument' introduced into the rectum not only caused intra-abdominal injuries, but completely transversed the abdomen, penetrated the diaphragm and pericardium, and entered the heart.

Case Report

A carpenter, aged 46 years, was admitted to hospital on 28 August 1954 at 10 p.m., complaining of severe abdominal pain and vomiting. He gave no account of how the illness started, and would only repeat that it came on suddenly during the late afternoon, and that he collapsed. His wife said that she returned from a shopping trip to find the house locked. After she had repeatedly called through the letter box, the patient eventually appeared, clad only in his socks, and opened the door. He looked very ill and then collapsed. His legs were soiled with faeces; the upstairs lavatory was soiled with faeces and blood. He vomited twice and although conscious gave no account of what had happened.

He was a very pale, well-built man, with a pulse rate of 130 per minute and blood pressure of 90/50 mm. Hg. He was distressed but rational. There were superficial abrasions over the front of the chest. There was no clinical abnormality in the chest, except that the apex beat was just outside the midclavicular line, and there was a systolic murmur conducted to the axilla. The abdomen was tender and rigid all over, and silent on auscultation. There was tenderness on rectal examination, which he resented, and slight blood staining of the examining finger. The only diagnosis which would fit the physical signs and the circumstances in which

he was found was a penetrating wound of the rectum, causing a haemoperitoneum. It was put to him that he might have accidentally sat on a spike of sorts, but this he denied, so that the diagnosis had to rest on circumstantial evidence.

After admission he was transfused with 3 pints of blood, but there was only a slight improvement in his condition. It was decided to perform a laparotomy without further delay, as there appeared to be persistent intraperitoneal bleeding.

Operation (4 *Hours after Admission*). Rectal examination under anaesthesia revealed a palpable wound in the anterior rectal wall. A laparotomy was carried out through a right paramedian incision. The peritoneal cavity contained about 2 pints of blood, which was removed. There was an oblique tear through the mesentery of the sigmoid colon, extending upwards into the peritoneum of the posterior abdominal wall. There was also a tear in the peritoneum on the left side at the brim of the pelvis, and from this a haemorrhagic track led down to the wound in the anterior wall of the rectum. Several bleeding points in the torn mesentery were ligatured. The peritoneum was repaired and a left iliac loop colostomy performed. There was no injury to the small intestine or its mesentery and no other injury could be seen higher up. There was no further bleeding. The wound was closed, with a drain down to the pelvis.

At the conclusion of the operation his general condition was slightly improved. The improvement was not maintained and his condition gradually deteriorated, in spite of further transfusion. He developed circulatory failure and died 11½ hours after admission.

Post-Mortem Examination

The body was 5′ 10″ tall and approximately 12 stone in weight. The only external marks were 10 very superficial small vertical abrasions on the front of the chest and upper abdomen. There was no bruising of the anal mucosa or the sphincter, and there were no marks on the peri-anal skin.

The wound in the anterior wall of the rectum was 6 cm. above the anal margin and 2·5 cm. in diameter. There was a tear in the left side of the pelvic peritoneum which had been sutured. The pelvic mesocolon was perforated, and had been sutured, and this wound extended vertically into the peritoneum of the posterior abdominal wall. A haemorrhagic track was found running upwards from this, passing behind the body of the pancreas and opening into the lesser sac. The track terminated in a tear (2·5 cm. in diameter) in the central tendon of the diaphragm, just to the left of the inferior vena cava, leading into the pericardial cavity.

The pericardium contained 8 oz. of fluid blood. There was a ragged tear (3 cm. long) in the wall of the right atrium. The edges were lightly adherent by pinkish-purple ante-mortem thrombus, but separate in the middle third. There were streaks of similar ante-mortem thrombus

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he was found was a penetrating wound of the rectum, causing a haemoperitoneum. It was put to him that he might have accidentally sat on a spike of sorts, but this he denied, so that the diagnosis had to rest on circumstantial evidence.

After admission he was transfused with 3 pints of blood, but there was only a slight improvement in his condition. It was decided to perform a laparotomy without further delay, as there appeared to be persistent intraperitoneal bleeding.

Operation (4 *Hours after Admission*). Rectal examination under anaesthesia revealed a palpable wound in the anterior rectal wall. A laparotomy was carried out through a right paramedian incision. The peritoneal cavity contained about 2 pints of blood, which was removed. There was an oblique tear through the mesentery of the sigmoid colon, extending upwards into the peritoneum of the posterior abdominal wall. There was also a tear in the peritoneum on the left side at the brim of the pelvis, and from this a haemorrhagic track led down to the wound in the anterior wall of the rectum. Several bleeding points in the torn mesentery were ligatured. The peritoneum was repaired and a left iliac loop colostomy performed. There was no injury to the small intestine or its mesentery and no other injury could be seen higher up. There was no further bleeding. The wound was closed, with a drain down to the pelvis.

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adhering to the visceral pericardium, mainly around the roots of the great vessels.

Inside the heart was a perforation (also 3 cm. long) in the inter-atrial septum just anterior to the closed foramen ovale. Adhering to its edges was a little ante-mortem thrombus, but it was widely patent.

Fig. 1. Photograph showing abdominal and thoracic viscera with a rod outlining the track made by the mop handle (inset).

The left pleural cavity contained 20 oz. of heavily blood-stained fluid, and the right 12 oz. There was an area of haemorrhage (10 x 6 cm.) in the left dome of the diaphragm extending from the wound already described, and there was some haemorrhage between the layers of the right pulmonary

ligament. Both lower lobes were partly collapsed and the lungs were moderately oedematous.

The heart was of normal size. Apart from the injuries described above there were no other signs of disease in the body. The distance between the anus and the right atrium was approximately 18 inches.

Discussion

From these findings it was deduced that an instrument over 18 inches long had entered through the anus and penetrated through the rectal wall as far as the heart. Absence of bruising around the anus suggested that the instrument must have been placed in the rectum before it penetrated further. A search was made for a possible instrument and a mop with a handle, 4 ft. long and 1 inch (2·4 cm.) in diameter and a rounded end, was found. The handle was then clean, but it was stated to have been soiled with faeces at the time of the accident.

The most likely mechanism for this injury is thought to be a fall, when the instrument was in the anus, so that the weight of the body forced the blunt-ended mop handle through the abdomen from the rectum to the heart. It is remarkable that after such an injury the patient could remove the instrument, walk downstairs, and live for 13 hours.

Summary

The case is reported of a wound of the heart caused by a blunt-ended mop handle which entered the body through the rectum.

We wish to thank Mr. C. P. Allen for permission to cite this case and also H. H. Kenshole, Esq., H. M. Coroner for the Metropolitan District of Essex, who held the subsequent inquest.

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Medicolegal Applications of Blood Grouping Tests*

A Tentative Supplementary Report* of the Committee on Medicolegal Problems, American Medical Association, prepared by Alexander S. Wiener, M.D., Brooklyn, N.Y., Ray D. Owen, Ph.D., Pasadena, Calif., Clyde Stormont, Ph.D., Davis, Calif., and Irving B. Wexler, M.D., Brooklyn, N.Y.

In 1952, a report[1] was prepared by the Committee on Medicolegal Problems of the American Medical Association describing the status at that time of the medicolegal application of blood-grouping tests for the exclusion of parentage and for the examination of stains of blood and secretions. The purpose of this supplementary report is to review further developments in the field during the period 1952—1955.

Blood Group Nomenclature

One of the most pressing problems at the present time is that of blood group nomenclature. This pertains particularly to the important Rh-Hr types, for which two principal methods of designation are in use, namely, the original Rh-Hr nomenclature and the C-D-E symbols. The submission to jurists of medicolegal reports by different experts with conflicting symbolisms cannot help but confuse them and shake their confidence in the blood tests. Moreover, reports in two different symbolisms require translation from one into the other when comparisons are to be made; there is always danger of error in this process. Therefore, the adoption of one uniform nomenclature for medicolegal reports is most desirable.

Fortunately, sufficient evidence is presently available to make possible a firm and unqualified recommendation. Although corresponding to the

* Before this report was placed in its present form, a preliminary draft was sent to 48 leading workers in the field of blood grouping. Responses have been received from: F. H. Allen, W. C. Boyd, G. J. Brancato, W. E. Briles, I. Davidsohn, W. Dameshek, L. A. Erf, S. T. Gibson, J. J. Griffitts, P. Levine, R. Murray, S. Raffel, W. D. Robinson, R. E. Rosenfield, J. B. Ross, S. B. Schatkin, M. Shapiro, R. T. Simmons, L. H. Snyder, W. H. Stone, H. H. Strandskov, L. N. Sussman, L. J. Unger, P. Vogel, M. M. Wintrobe, and E. Witebsky. An effort has been made to take all the comments received into account and to include all pertinent facts when preparing this final version of the report. Needless to say, this does not imply that the report has the unanimous endorsement of all those who responded. There was a wide diversity of opinions expressed in the replies, ranging from enthusiastic approval to violent disapproval. In fact, it was this very state of confusion among workers in the field that originally made it necessary to prepare this report. In arriving at its conclusions, this fact-finding committee has necessarily excluded all comments that, in its opinion, were incorrect or unsound and has included only those that the committee believes to have a firm scientific basis.

two systems of designating the Rh-Hr types there are two conflicting genetic theories, the crux of the problem is actually on a serologic level. It is most important to distinguish clearly between agglutinogens and their serologic attributes, the blood factors.[2] An agglutinogen may be defined as a substance present on the surface of the red blood cells that is identified by certain agglutination reactions with diagnostic reagents. The blood factors, on the other hand, are those attributes of the agglutinogen molecule (perhaps an arrangement of polar groups, electric charges, and other configurational details on the surface of the molecule) that enable it to combine with antibodies. In general, each agglutinogen is characterized by multiple blood factors. The C-D-E notations for the Rh-Hr types make no allowance for the difference between a blood factor and an agglutinogen and incorporate the tacit, incorrect assumption that every agglutinogen has but a single corresponding antibody. This has led to a number of paradoxes, which have been described in detail elsewhere[3] and need not be elaborated here. Indeed, much of the evidence that has accumulated regarding the serology and genetics of the Rh-Hr types refutes the interpretation implicit in the C-D-E notations. Furthermore, there has been a noticeable attempt to force the facts to fit the C-D-E hypothesis. On the other hand, the original Rh-Hr nomenclature presents the data objectively, without committing the user to a preconceived interpretation, which at times may be inconsistent with the actual observations. Under these circumstances, the committee has been obliged to adopt the original Rh-Hr nomenclature as the sole nomenclature in the preparation of this report. This committee, therefore, recommends that the C-D-E notations for the Rh-Hr types be discarded and that, in approved medicolegal reports, unless and until some other convention is agreed upon, the original Rh-Hr nomenclature be retained as the sole nomenclature for this blood group system (Table 1).

In any blood group system, and the Rh-Hr system in particular, one must carefully distinguish among agglutinogens, blood factors, and genes. To avoid confusion among these three levels, appropriate provisions must be made in the symbols. Therefore, it is suggested that regular-face type be used for agglutinogens and phenotypes, bold-face type for blood factors and their corresponding antibodies, and italic type for genes and genotypes. This is in keeping with accepted genetic usage. This recommendation is approved by this committee and has been adopted for the purpose of this report. For purposes of illustration and for its practical value, the heredity of the Rh-Hr types will be briefly reviewed, using the recommended nomenclature.

At the present time, four principal Rh blood factors (**Rh_0**, **rh′**, **rh″**, and **rh^w**) and three principal Hr factors (**hr′**, **hr″**, and **hr**) are recognized. However, antiserums for only four of these seven factors are readily available

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Table 1: Rh-Hr Phenotypes and Genotypes

2 *Rh Phenotypes*			12 *Rh Phenotypes*					28 *Rh-Hr Phenotypes*					
Designations	*Approximate Frequencies in New York City Whites* %	*Reaction with Anti-*Rh_0 *(or Anti-rhesus)*	*Designation*†	*Approximate Frequencies in New York City Whites* %‡	*Reaction with* Anti-**rh′**	Anti-**rh″**	Anti-rh^w	*Designation*	*Approximate Frequencies in New York City Whites* %§	*Reaction with* Anti-**hr′**	Anti-**hr″**	Anti-**hr**	*55 Genotypes**
Rh negative	15	—	rh	14·4	—	—	—	rh	14·4	+	+	+	*rr*
			rh′	0·46†	+	—	—	rh′rh	0·46	+	+	+	*r′r*
								rh′rh′	0·0036	—	+	—	*r′r′*
			rh'^w	0·004	+	—	+	rh'^wrh	0·004	+	+	+	r'^wr
								rh'^wrh'	0·00006	—	+	—	r'^wr' or $r'^wr'^w$
			rh″	0·38	—	+	—	rh″rh	0·38	+	+	+	*r″r*
								rh″rh″	0·0025	+	—	—	*r″r″*
			rh_y	0·01	+	+	—	rh′rh″	0·006	+	+	—	*r′r″*
								rh_yrh	0·008	+	+	+	r^yr
								rh_yrh'	0·0001	—	+	—	r^yr'
								rh_yrh''	0·0001	+	—	—	r^yr''
								rh_yrh_y	0·000001	—	—	—	r^yr^y
			rh_y^w	0·00005	+	+	+	rh'^wrh''	0·00005	+	+	—	r'^wr''
								rh_y^wrh'	0·000001	—	+	—	r'^wr^y

			rh_y	0·00005				$rh_y^w rh'$	0·000001	—	+	—	$r'^w r^y$
Rh positive	85	+	Rh_0	2·1	—	—	—	Rh_0	2·1	+	+	+	R^0R^0 or R^0r
			Rh_1	50·7	+	—	—	Rh_1rh	33·4	+	+	+	R^1r, R^1R^0, or R^0r'
								Rh_1Rh_1	17·3	—	+	—	R^1R^1 or R^1r'
			Rh_1^w	3·3	+	—	+	Rh_1^wrh	1·6	+	+	+	$R^{1w}r$, $R^{1w}R^0$, or $R^0r'^w$
								$Rh_1^wRh_1$	1·7	—	+	—	$R^{1w}R^1$, $R^1r'^w$, $R^{1w}r'$, $R^{1w}R^{1w}$, or $R^{1w}r'^w$
			Rh_2	14·6	—	+	—	Rh_2rh	12·2	+	+	+	R^2r, R^2R^0, or R^0r''
								Rh_2Rh_2	2·4	+	—	—	R^2R^2 or R^2r''
			Rh_z	13·4	+	+	—	Rh_1Rh_2	12·9	+	+	—	R^1R^2, R^1r'', or R^2r'
								Rh_zrh	0·2	+	+	+	R^zr, R^zR^0, or R^0r^y
								Rh_zRh_1	0·2	—	+	—	R^zR^1, R^zr', or R^1r^y
								Rh_zRh_2	0·07	+	—	—	R^zR^2, R^zr'', or R^2r^y
								Rh_zRh_z	0·0004	—	—	—	R^zR^z or R^zr^y
			Rh_z^w	0·6	+	+	+	$Rh_1^wRh_2$	0·6	+	+	—	$R^{1w}R^2$, $R^{1w}r''$, or $R^2r'^w$
								$Rh_z^wRh_1$	0·008	—	+	-	$R^{1w}R^z$, $R^{1w}r^y$, or $R^zr'^w$

* This table does not include hypothetical genes R^{zw} and r^{yw}, which, if they exist at all, are very rare.

† In this table Rh_1 is used as a short designation for Rh_0', Rh_2 is short for Rh_0'', rh_y is short for rh′ ″, and Rh_z is short for Rh_0' ″.

‡ The reduction in the frequency of type rh′ as compared with that given in earlier charts can be attributed to recognition of bloods of type Rh_1 (containing Rh_0 variant), which are now included in type Rh_1 instead of rh′. The agglutinogens $\mathfrak{R}h_0$, $\mathfrak{R}h_1$ and $\mathfrak{R}h_2$ and their corresponding genes $\mathfrak{R}^0$, $\mathfrak{R}^1$ and $\mathfrak{R}^2$ are not given here because this would serve unnecessarily to complicate the chart, by increasing the number of possible genotypes to 91. Also no attempt is made to include certain rare exceptional bloods, such as those lacking both factors **rh′** and **hr′** and/or lacking both **rh″** and **hr″**.

§ Based on the estimated gene frequencies, $r=0·38$, $r'=0·006$, $r''=0·005$, $r^y=0·0001$, $r'^w=0·00005$, $R^0=0·027$, $R^1=0·41$, $R^2=0·15$, $R^z=0·002$ and $R^{1w}=0·02$.

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for routine clinical and medicolegal work, namely, anti-**Rh_0**, anti-**rh′**, anti-**rh″**, and anti-**hr′**. Therefore, this discussion will be confined to the results obtained using these four antiserums. Ample evidence is now available that there are eight principal alleles determining the heredity of the Rh-Hr types, as distinguished by these four reagents. Table 2 lists these alleles and their corresponding agglutinogens, as well as the blood factors

Table 2: Eight 'Standard' Allelic Genes and Their Corresponding Agglutinogens

Genes	*Corresponding Agglutinogens*	*Blood Factors Characterizing Each Agglutinogen**
r	rh′	**hr′**
r'	rh′	**rh′**
r''	rh″	**rh″** and **hr′**
r^y	rh_y	**rh′** and **rh″**
R^0	Rh_0	**Rh_0** and **hr′**
R^1	Rh_1	**Rh_0** and **rh′**
R^2	Rh_2	**Rh_0**, **rh″**, and **hr′**
R^z	Rh_z	**Rh_0**, **rh′**, and **rh″**

* Taking into account only reactions with serums anti-**Rh_0**, anti-**rh′**, anti-**rh″**, and anti-**hr′**.

that characterize each agglutinogen. Table 3 gives the various reactions that may be encountered in actual tests carried out with the four Rh-Hr antiserums. It will be seen that there are 12 possible phenotypes, to which there correspond 36 theoretically possible genotypes. With this basic information, it is not difficult to determine which Rh-Hr types can occur and which cannot for any given mating.

Taking into account the 12 possible phenotypes, there are 78 matings to be considered. (When the reactions of anti-**hr″** serums are also taken into account, the number of phenotypes is increased to 18 and the number of matings to 171. A table listing these 171 matings has been published by Wiener.[4]) A table listing the 78 matings would require more than two pages. Boyd[5] has devised a compact method of presenting the various matings in the form of a '12 by 12' table, and Table 4 was drawn up after examining the similar table prepared by Boyd. It will be noticed that the phenotypes have been numbered, but these numbers are not intended to be used as a new nomenclature for the Rh-Hr types. The phenotype numbers are used merely for compactness in presenting the table, and the key to the numbers is given at the top and side of the chart. For example, in the mating $Rh_2 \times Rh_2$ phenotype numbers 2, **3**, 5, **6**, 8, **9**, 11 and **12**

appear in the box, which means that parents both of type Rh_2 cannot have children of types rh′rh, rh′rh′, rh′rh″, rh_yrh', Rh_1rh, Rh_1Rh_1, Rh_1Rh_2, or Rh_2Rh_1. This is readily verified with the aid of Tables 2 and 3, since both parents of type Rh_2 can be carriers of genes R^2, R^0, r'', or r_s, and not of genes R^1, R^z, r', or r^y, so that all children with phenotypes in which one or more of the latter four genes must be present are excluded. The numbers in bold-face type indicate phenotypes that, if present in the child, exclude maternity. Thus, a type Rh_2 mother cannot have a child of type rh′rh′, rh_yrh', Rh_1Rh_1, or Rh_2Rh_1.

Table 3: Rh-Hr Phenotypes and Corresponding Genotypes

Phenotype	*Reaction with Serums*				*Corresponding Genotypes*
	Anti-$\mathbf{Rh_0}$	Anti-**rh′**	Anti-**rh″**	Anti-**hr′**	
rh	—	—	—	+	rr
rh′rh ..	—	+	—	+	$r'r$
rh′rh′ ..	—	+	—	—	$r'r'$
rh″.. ..	—	—	+	+	$r''r''$ and $r''r$
rh′rh″ ..	—	+	+	+	$r'r''$, r^yr, and r^yr''
rh_yrh' ..	—	+	+	—	r^yr' and r^yr^y
Rh_0 ..	+	—	—	+	R^0R^0 and R^0r
Rh_1rh ..	+	+	—	+	R^1r, R^1R^0, and R^0r'
Rh_1Rh_1 ..	+	+	—	—	R^1R^1 and R^1r'
Rh_2 ..	+	—	+	+	R^2R^2, R^2r'', R^2r, R^2R^0, and R^0r''
Rh_1Rh_2 ..	+	+	+	+	R^1R^2, R^1r'', R^2r', R^2r, R^2R^0, R^2R^2, R^2r'', R^0r^y, and R^2r^y
Rh_2Rh_1 ..	+	+	+	—	R^2r', R^2r^y, R^2R^1, R^2R^2, and R^1r^y

Use of Newer Blood Factors in Cases of Disputed Parentage

With the development of new methods of detecting antibodies, namely, the conglutination test, the antiglobulin test, and tests using red blood cells treated with proteolytic enzymes, it has been possible to resolve clinical problems that previously were insoluble. Thus, certain obscure hemolytic transfusion reactions have been shown to be due to isosensitization to some blood factor of the transfused red blood cells, and the occurrence of hemolytic disease of fetus and newborn infant when the mother is Rh positive has been explained on a similar basis. While the situation is not quite as complex as in cattle blood groups, up to the present time as many as 30 different blood factors have been identified in human blood. For ordinary clinical purposes only three of the human blood factors are numerically

Table 4: Exclusion of Parentage by Rh-Hr Tests

Phenotype of Putative Mother	*Phenotype of Putative Father*											
	1 rh	2 rh′rh	3 rh′rh′	4 rh″	5 rh′rh″	6 rh_yrh'	7 Rh_0	8 Rh_1rh	9 Rh_1Rh_1	10 Rh_2	11 Rh_1Rh_2	12 Rh_2Rh_1
1. rh′ ..	2, **3,** 4, 5, **6,** 7, 8, **9,** 10, 11, **12**	**3,** 4, 5, **6,** 7, 8, **9,** 10, 11, **12**	1, **3,** 4, 5, **6,** 7, 8, **9,** 10, 11, **12**	2, **3,** 5, **6,** 7, 8, **9,** 10, 11, **12**	**3, 6,** 7, 8, **9,** 10, 11, **12**	1, **3,** 4, **6,** 7, 8, **9,** 10, 11, **12**	2, **3,** 4, 5, **6,** 8, **9,** 10, 11, **12**	**3,** 4, 5, **6, 9,** 10, 11, **12**	1, **3,** 4, 5, **6,** 7, **9,** 10, 11, **12**	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9, 12**	1, **3,** 4, **6,** 7, **9,** 10, **12**
2. rh′rh ..	3, 4, 5, 6, 7, 8, 9, 10, 11, 12	4, 5, 6, 7, 8, 9, 10, 11, 12	1, 4, 5, 6, 7, 8, 9, 10, 11, 12	3, 6, 7, 8, 9, 10, 11, 12	7, 8, 9, 10, 11, 12	1, 4, 7, 8, 9, 10, 11, 12	3, 4, 5, 6, 9, 10, 11, 12	4, 5, 6, 10, 11, 12	1, 4, 5, 6, 7, 10, 11, 12	3, 6, 9, 12	None	1, 4, 7, 10
3. rh′rh′	**1,** 3, **4,** 5, 6, **7,** 8, 9, **10,** 11, 12	**1, 4,** 5, 6, **7,** 8, 9, **10,** 11, 12	**1,** 2, **4,** 5, 6, **7,** 8, 9, **10,** 11, 12	**1,** 3, **4,** 6, **7,** 8, 9, **10,** 11, 12	**1, 4, 7,** 8, 9, **10,** 11, 12	**1,** 2, **4,** 5, **7,** 8, 9, **10,** 11, 12	**1,** 3, **4,** 5, 6, **7,** 9, **10,** 11, 12	**1, 4,** 5, 6, **7, 10,** 11, 12	**1,** 2, **4,** 5, 6, **7,** 8, **10,** 11, 12	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, **7,** 8, **10,** 11
4. rh″ ..	2, **3,** 5, **6,** 7, 8, **9,** 10, 11, **12**	**3, 6,** 7, 8, **9,** 10, 11, **12**	1, **3,** 4, **6,** 7, 8, **9,** 10, 11, **12**	2, **3,** 5, **6,** 7, 8, **9,** 10, 11, **12**	**3, 6,** 7, 8, **9,** 10, 11, **12**	1, **3,** 4, **6,** 7, 8, **9,** 10, 11, **12**	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9, 12**	1, **3,** 4, **6,** 7, **9,** 10, **12**	2, **3,** 5, **6,** 8, **9,** 11, **12**	3, **6, 9, 12**	1, **3,** 4, **6,** 7, **9,** 10, **12**
5. rh′rh″	3, 6, 7, 8, 9, 10, 11, 12	7, 8, 9, 10, 11, 12	1, 4, 7, 8, 9, 10, 11, 12	3, 6, 7, 8, 9, 10, 11, 12	7, 8, 9, 10, 11, 12	1, 4, 7, 8, 9, 10, 11, 12	3, 6, 9, 12	None	1, 4, 7, 10	3, 6, 9, 12	None	1, 4, 7, 10
6. rh_yrh'	**1,** 3, **4,** 6, **7,** 8, 9, **10,** 11, 12	**1, 4, 7,** 8, 9, **10,** 11, 12	**1,** 2, **4,** 5, **7,** 8, 9, **10,** 11, 12	**1,** 3, **4,** 6, **7,** 8, 9, **10,** 11, 12	**1, 4, 7,** 8, 9, **10,** 11, 12	**1,** 2, **4,** 5, **7,** 8, 9, **10,** 11, 12	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, **7,** 8, **10,** 11	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, **7,** 8, **10,** 11

Table 4: Exclusion of Parentage by Rh-Hr Tests (*Continued*)

Phenotype of Putative Mother	*Phenotype of Putative Father*											
	1 rh	2 rh′rh	3 rh′rh′	4 rh″	5 rh′rh″	6 rh_yrh'	7 Rh_0	8 Rh_1rh	9 Rh_1Rh_1	10 Rh_2	11 Rh_1Rh_2	12 Rh_2Rh_1
7. Rh_0 ..	2, **3,** 4, 5, **6,** 8, **9,** 10, 11, **12**	**3,** 4, 5, **6, 9,** 10, 11, **12**	1, **3,** 4, 5, **6,** 7, **9,** 10, 11, **12**	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9, 12**	1, **3,** 4, **6,** 7, **9,** 10, **12**	2, **3,** 4, 5, **6,** 8, **9,** 10, 11, **12**	**3,** 4, 5, **6, 9,** 10, 11, **12**	1, **3,** 4, 5, **6,** 7, **9,** 10, 11, **12**	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9, 12**	1, **3,** 4, **6** 7, **9,** 10, **12**
8. Rh_1rh	3, 4, 5, 6, 9, 10, 11, 12	4, 5, 6, 10, 11, 12	1, 4, 5, 6, 7, 10, 11, 12	3, 6, 9, 12	None	1, 4, 7, 10	3, 4, 5, 6, 9, 10, 11, 12	4, 5, 6, 10, 11, 12	1, 4, 5, 6, 7, 10, 11, 12	3, 6, 9, 12	None	1, 4, 7, 10
9. Rh_1Rh_1	**1,** 3, **4,** 5, 6, **7,** 9, **10,** 11, 12	**1, 4,** 5, 6, **7, 10,** 11, 12	**1,** 2, **4,** 5, 6, **7,** 8, **10,** 11, 12	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, **7,** 8, **10,** 11	**1,** 3, **4,** 5, 6, **7,** 9, **10,** 11, 12	**1, 4,** 5, 6, **7, 10,** 11, 12	**1,** 2, **4,** 5, 6, **7,** 8 **10,** 11, 12	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, **7,** 8, **10,** 11
10. Rh_2 ..	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9, 12**	1, **3,** 4, **6,** 7, **9,** 10, **12**	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9, 12**	1, **3,** 4, **6,** 7, **9,** 10, **12**	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9, 12**	1, **3,** 4, **6,** 7, **9,** 10, **12**	2, **3,** 5, **6,** 8, **9,** 11, **12**	**3, 6, 9, 12**	1, **3,** 4, **6,** 7, **9,** 10, **12**
11. Rh_1Rh_2	3, 6, 9, 12	None	1, 4, 7, 10	3, 6, 9, 12	None	1, 4, 7, 10	3, 6, 9, 12	None	1, 4, 7, 10	3, 6, 9, 12	None	1, 4, 7, 10
12. Rh_2Rh_1	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, **7,** 8, **10,** 11	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, **7,** 8, **10,** 11	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, **7,** 8, **10,** 11	**1,** 3, **4,** 6, **7,** 9, **10,** 12	**1, 4, 7, 10**	**1,** 2, **4,** 5, **7,** 8, **10,** 11

* To understand and use this chart quickly and easily, the phenotype names and numbers should be used in conjunction with the table of Rh-Hr reactions (Table 3). 1. Determine the phenotypes of the blood of the mother, putative father, and the child or children using potent and specific anti-**Rh_0**, anti-**rh′**, anti-**rh″**, and anti-**hr′** serums, including appropriate controls. 2. Assign the corresponding phenotype symbol and number to each individual tested. 3. Find the assigned number of the mother and father on the side and top columns of the table and locate the box at which both intersect. 4. The child or children are not possible from this mating if their assigned numbers appear in this box. 5. Phenotype numbers given in boldface represent children for whom maternity is excluded.

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important, namely, **A**, **B**, and **Rh_0**; in blood transfusion practice reliance is placed on the cross matching test to detect those rare cases (Table 5) of isosensitization to other blood factors.

Studies on the distribution and heredity of the agglutinogens and blood factors have shown that certain of them are related to one another so as to form systems. Other factors have the peculiarity that blood lacking the factor or blood possessing the factor is extremely rare. Table 5 summarizes the present status of knowledge of the human blood groups, with special reference to their role in blood transfusion. In medicolegal cases, when blood tests are carried out in problems of disputed parentage, the examination is usually limited to the A-B-O, M-N, and Rh-Hr systems. Moreover, the tests are further limited to particular factors of these three systems. In the preceding report of 1952, it was recommended that the medicolegal application of the blood test should not be extended further for the time being. For reasons pointed out below, it is still premature to apply these newer tests routinely in medicolegal cases, though there might be special circumstances when their use would be permissible.

A-B-O System. For general medicolegal purposes, the blood tests should be restricted to the factors **A** and **B** and to the four blood groups O, A, B, and AB. The tests should include examination of the serum (or plasma) of each individual for its isoagglutinin content, as well as tests on the red blood cells with anti-**A** and anti-**B** serums.[6] Tests for the subgroups of A are not sufficiently reliable for medicolegal use. The serologic distinction between agglutinogens A_1 and A_2 is not sharp, so that the diagnosis is not infrequently difficult to make, especially in newborn babies and in individuals of group AB. Moreover, intermediate forms exist between A_1 and A_2. At any rate, the chances of excluding paternity for a falsely accused man, which is about 18·5% using factors **A** and **B**, would be increased by only 1 or 2% were the subgroups of A applied. Therefore, this committee finds that tests for the subgroups of A remain too unreliable for general medicolegal use.

M-N-S System. For medicolegal purposes the paternity blood tests are preferably restricted to the factors **M** and **N**. Antiserums for these factors are prepared in rabbits, and from most of the antiserums satisfactory reagents can be prepared that give reactions of high avidity and specificity. The reagents, particularly for factor **N**, are generally of low titer, but this does not appear to interfere with the reliability of the results. Satisfactory anti-**N** reagents can also be prepared from the seeds of *Vicia graminae*, which may be used if desired for purposes of confirmation.[7] Under no circumstances should the diagnosis be based solely on tests with such extracts. Tests on tens of thousands of families and mother-child combinations have proved that exclusion of paternity based on the M-N types are as reliable as exclusions based on the A-B-O tests.

Reagents for factor **S** are obtained from human individuals sensitized by transfusion or pregnancy. While the titer of these reagents is generally low, the reactions are of high avidity and specificity like the tests for **M** and **N**. However, the number of studies to date on the **S** factor is small, because of the scarcity of this reagent, which, unlike anti-**M** and anti-**N** cannot, at present, be produced at will. Therefore, the medicolegal application of the **S** test should be limited to selected cases, and exclusions of parentage based on this test alone should be qualified by pointing out that the statistical basis for this test is still incomplete, though there is no reason to doubt its reliability. Reagents for anti-**s** are even rarer than anti-**S**. Moreover, the anti-**s** reagents available up to the present had to be used with the antiglobulin technique, and the results were reproducible only with difficulty. For these reasons, the medicolegal application of factor **s** is not recommended for the present. It is hoped that the technique of the test for factor **s** will be perfected, since the combined tests for **S** and **s** will increase substantially the chances of excluding paternity. The addition of anti-**S** alone adds about 40% as much as the addition of both anti-**S** and anti-**s**. Antiserums for factors Hunter and Henshaw (Table 5) are still in limited use, so that such tests are not yet available for medicolegal purposes. Recent work indicates that factor **U** is related to the M-N-S system, but, because of the almost universal distribution of this factor, it has little if any medicolegal importance.

Rh-Hr System. As has already been pointed out, in medicolegal cases the paternity blood tests are generally limited to the factors **Rh_0**, **rh′**, **rh″**, and **hr′**. Actually many more Rh-Hr factors have been found. Of all the Rh-Hr factors known, the most important are the four Rh factors **Rh_0**, **rh′**, **rh″**, and **rh^w** and the three Hr factors **hr′**, **hr″**, and **hr**. The reactions of these seven antiserums and the types they determine are given in Table 1.

As can be seen from Table 1, as many as 28 sharply defined Rh-Hr phenotypes can be differentiated with the aid of the 7 Rh-Hr serums, corresponding to which there are 55 genotypes theoretically possible. This does not take into account the additional phenotypes and genotypes resulting from the existence of **Rh_0** variants and certain other rare Rh-Hr agglutinogens. In practice the number of phenotypes encountered will be considerably smaller, because many of the phenotypes are rare. Thus if only those phenotypes that have an incidence of 1% or higher are counted, there are only 9 or 10 phenotypes among those theoretically possible that are apt to be encountered in tests on a short series of individuals. The medicolegal expert must also be aware of the existence of rare individuals with an agglutinogen having the factor **Rh_0** but lacking both factors of each of the two pairs **rh′-hr′** and **rh″-hr″**. Such bloods have been labeled as 'deletions,' but a more conservative designation for this type is Rh_0^x with $R^{ox}R^{ox}$ as the corresponding genotype. The existence of these rare agglutinogens

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Table 5: Blood Groups in Man: Role in Blood Transfusion

System	*Blood Factors*	*Number of Identifiable Types*	*Isoantibodies*		*Role in Causing Transfusion Hemolysis*
			Natural	*Isoimmune*	
A-B-O	**A**, **B**, **C**, **O**, and variants such as $\mathbf{A_1}$, $\mathbf{A_2}$, $\mathbf{A_{1,2}}$, and $\mathbf{A_3}$	6 major groups and subgroups: O, A_1, A_2, B, A_1B, A_2B; also rarer subgroups as A_3, A_3B	Regularly present except during neonatal period	Common	Most important
M-N-S	**M**, **N**, **S**, **s**, **Hu** (Hunter), **He** (Henshaw), **U**, and variants such as $\mathbf{M_2}$, $\mathbf{N_2}$	6 major types using the more readily available serums anti-**M**, anti-**N**, and anti-**S**	Rare	Rare	Has caused hemolysis in rare cases
Rh-Hr	$\mathbf{Rh_0}$, **rh′**, **rh″**, $\mathbf{rh^w}$, **hr′**, **hr″**, **hr**, $\mathbf{rh^v}$, and variants such as $\mathbf{Rh_0}$	28 major types, but only 9 have frequencies greater than 1%	A few questionable cases reported	Common	Most frequent cause of intragroup transfusion hemolysis
P	**P** and variants, $\mathbf{Tj^a}$	2 major types	Infrequent	Very rare	Rare cause of reactions
K-k	**K** and **k**	3	None reported	Occasional	Rare cause of hemolysis in isosensitized patients
Lutheran	**Lu**	2	None reported	1 case	Very rare cause of hemolysis in isosensitized patients

Table 5: Blood Groups in Man: Role in Blood Transfusion (*Continued*)

System	*Blood Factors*	*Number of Identifiable Types*	*Isoantibodies*		*Role in Causing Transfusion Hemolysis*
			Natural	*Isoimmune*	
Duffy (Fy)	**F** and f	2 major types, because anti-f serum is not available	None reported	Several cases	Rare cause of hemolysis
Lewis (Le)	**Le**^{a} and **Le**^{b}	3 (?)	Infrequent	None found	Plays no role in transfusion hemolysis. **Le**^{a} factor reciprocally related to secretor trait
Kidd (Jk)	**J** and j	2 major types, because anti-j serum is not available	None reported	Few cases	Rare cause of hemolysis
"*Private*" blood types* Rare blood factors ..	**Levay**, **Gr** (Graydon), **Mi**^{a} (Miltenberger), **Becker**, **Be**^{a} (Berrens), **Ca** (Cavaliere), **Wr**^{a}, Diego‡	Positively reacting bloods are very rare	None reported	Very rare	Very rare cause of hemolysis
Factors with almost universal distribution†	**Vel** and **I**	Negatively reacting bloods are very rare	None reported	1 case anti-**I**; 2 cases anti-**Vel**	Very rare cause of transfusion hemolysis

* In the case of the rare blood factors, other individuals possessing such factors are most easily found by testing relatives of the proband, the person in whose blood the factor was first found. Similarly, individuals lacking factors of almost universal distribution are to be found most readily among relatives of the proband. The hereditary nature of the blood factors, in general, is most noticeable in the case of very rare blood types, and Levine has, therefore, designated them as "private" blood types to indicate their restriction to certain rare families.

† Factor **k** of the K-k system, **Tj**^{a} of the P system, and **U** of the M-N-S system could also be included under this classification.

‡ Diego factor is common, however, in Amerindians.

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affects the validity of the rule that an **rh**′-negative parent cannot have an **hr**′-negative child and an **hr**′-negative parent cannot have an **rh**′-negative child. For example, if the putative father belongs to type Rh_1Rh_1 and the child to type Rh_2, paternity is not necessarily excluded, provided the man's genotype is R^1R^{ox}, and the baby's genotype R^2R^{ox}. Therefore, such exclusions of paternity call for some mild qualification. No qualification is needed when the rule is recorded as: An **rh**′-negative parent lacking the $\mathbf{Rh_0}$ factor cannot have an **hr**′-negative child, and an **hr**′-negative parent cannot have an **rh**′-negative child lacking the $\mathbf{Rh_0}$ factor.

The availability of anti-$\mathbf{rh^w}$ serum further increases the chances of excluding paternity. Aside from the fact that the $\mathbf{rh^w}$ factor cannot appear in a child unless present in one or both parents, particular combinations exclude paternity in certain instances where no decision would be possible without the aid of this antiserum. For example, a parent of type $Rh_1{}^wrh$ cannot have a child of type Rh_1Rh_1, and a parent of type Rh_1Rh_1 cannot have a child of type $Rh_1{}^wrh$.

The use of anti-**hr** serum (Table 1) may help resolve certain problems of disputed parentage. The recently discovered factor **hr** has no Rh factor counterpart, thus differing from factors **hr**′ and **hr**″, which have the counterparts **rh**′ and **rh**″ respectively. With possible rare exceptions, the factor **hr** has been found present in the agglutinogens determined by genes r and R^o, and absent from the agglutinogens determined by the genes r', r'', r^y, R^1, R^2, and R^z. When a putative parent belongs to type Rh_z (blood with the three factors $\mathbf{Rh_0}$, **rh**′, and **rh**″), and the child to type rh or Rh_0, or vice versa, parentage is excluded, provided it can be shown that the individual of type Rh_z is not carrying the genes R^z or r^y. This may be possible with the aid of anti-**hr** serum, namely, if it can be shown that the blood has factors **hr**′ and **hr**″ but lacks the **hr** factor (Table 1).

Recent work has revealed the existence of still other rare factors belonging to the Rh-Hr system, notably, factor **V**, often found associated with the genes r and R^o, especially in Negroes. Obviously, the medicolegal application of such factors is premature. Indeed, because of the lack of availability of adequate amounts of suitable antiserums and the limited knowledge due to the small number of investigations carried out to date, the routine use in medicolegal work of factors $\mathbf{rh^w}$, **hr**″, and **hr** is inadvisable. This committee recommends, therefore, that for routine medicolegal work, for the present, the Rh-Hr tests be limited to the factors $\mathbf{Rh_0}$, **rh**′, **rh**″, and **hr**′.

P System. The antiserums available for use have been mostly of human origin and contain irregular agglutinins giving most distinct reactions at low temperature. Due to the presence in such serums of interfering nonspecific cold agglutinins, and the low titer of these reagents, the results of such tests are not sufficiently reliable for medicolegal purposes. This

committee, therefore, recommends that the P tests should not be used in routine medicolegal practice. Recent work indicates that the high incidence factor **Tj**^{a} is related to the P system. It should also be mentioned that the so-called **Q** factor of Japanese investigators is probably identical with factor **P**.

Lewis System. Knowledge of the Lewis system is still in a confused state due to the lack of potent and specific reagents. The only reagents available appear to be natural irregular isoagglutinins encountered in rare human serums, which react best at low temperature and rarely have a titre exceeding 2 to 4 units. Due to the presence of interfering cold agglutinins, especially anti-**O** in many anti-**Le**^{b} serums, these tests are not sufficiently reliable for medicolegal use.

Kell System. Many additional examples of anti-**K** serum have been encountered during recent years, and all have similar properties, in that they react best by the conglutination and antiglobulin technique. Therefore, these reagents cannot be used for typing blood with red blood cells already coated by antibody globulin, as in patients with hemolytic disease of the fetus or newborn infant or acquired hemolytic anemia. Moreover, the agglutinates are somewhat more fragile than in Rh-Hr reactions, presumably due to the smaller number of antigenic loci on the discoplasm, so that the reactions are not always readily reproducible. This difficulty in technique has apparently discouraged extensive investigation of this system. The use of the rare anti-**k** serum adds little information, since only one out of approximately 500 bloods fails to clump in such antiserums. For these reasons, as well as the small number of family studies carried out to date, this committee recommends that the K-k tests should not be used in routine medicolegal practice but be reserved for special cases only.

Duffy and Kidd Systems. The Duffy and Kidd systems are grouped together because of the similarity in behaviour of the available reagents. These reagents give the most distinct reactions only by the antiglobulin method, and the agglutinates are even more fragile than the Kell tests. Because of the difficulty in reproducing the reactions, and the small number of family studies carried out to date, the Duffy and Kidd tests are not suitable for routine medicolegal purposes. Therefore, this committee recommends that tests for factors **F** (or **Fy**^{a}) and **J** (or **Jk**^{a}) should not be carried out in routine medicolegal practice. However, there are some experts who feel that these tests are sufficiently reliable for medicolegal use.

The so-called family, or private, factors (Table 5) have little or no value in medicolegal practice, because of their rarity in the general population.

Blood Tests as Circumstantial Evidence of Parentage)

The expert in the field of blood grouping is accustomed to think of blood tests as a method of establishing non-parentage only. When the blood types of the child match genetically with those of the supposed

parents this is considered an inconclusive finding because of the possibility of coincidence. However, with the increase in the number of detectable blood factors and the number of blood group systems whose mode of inheritance is known, the possibility suggests itself of using blood tests as circumstantial evidence of paternity or maternity. 'Assignment' of parents is occasionally possible in disputed parentage cases involving cattle.[8]

In most cases, despite the combined use of A-B-O, M-N, and Rh-Hr tests, such circumstantial evidence of parentage will not be strong. For example, if both putative parents and the child belong to type O M Rh_1rh, there is still a substantial chance of coincidence. Exactly where to draw the line is difficult to state. This committee feels, therefore, that the routine use of blood tests as circumstantial evidence of paternity is not advisable in routine medicolegal practice. In fact, attempts to apply blood tests in this way carry the danger that they may serve to confuse the issue and cause the court to discredit the valuable blood tests altogether.

On the other hand, there are certain unusual cases that cannot be ignored, where the use of blood tests as circumstantial evidence of paternity or maternity appears to be justified. For example, if the putative father belongs to type rh″, the mother to type rh, and the child to type rh″, the conscientious examiner will feel obligated to call this unusual circumstance to the attention of the court. Since rh″ occurs in less than 0·5% of the population, the coincidental presence of this type in the child and the accused man is circumstantial evidence of paternity. However, this committee feels that such an observation should not be included in the official report but should be mentioned and explained in a covering letter accompanying the report. Other situations in which the results of blood tests may perhaps be considered circumstantial evidence of paternity are cases where the child and accused man both have the factor $\mathbf{rh^x}$ or $\mathbf{rh^w}$, lacking from the blood of the mother. As another example may be cited the instance where the baby and putative father both are Kell positive and group B, while the mother is Kell negative and group O.

The committee feels that the use of blood tests as circumstantial evidence of parentage is justified only in selected unusual cases. In such instances, the observations of the examiner should not be incorporated in the formal report but should be pointed out in a covering letter accompanying the report. All tests should be performed and interpreted only by experts in the field of blood grouping.

Model Blood-Grouping Law

To further the medicolegal application of blood-grouping tests in cases of disputed paternity, the passage of state laws authorizing courts to order these tests appears to be necessary. At the time when the previous report of this committee was published, laws had been passed in the states of

Maine, Maryland, New Jersey, New York, North Carolina, Ohio, Pennsylvania, South Dakota and Wisconsin. During the past three years three more states have been added to the list, namely, Massachusetts, Minnesota and New Hampshire. The main obstacle to the adoption of similar laws by all states is the dearth of qualified experts specializing in the field of blood grouping. Even among workers in the field, unnecessary controversy regarding nomenclature certainly has not helped to instill confidence in the tests among lawyers and jurists. From the point of view of the legislator and jurist, the wider medicolegal application of blood tests may be fostered by the availability of a model blood-test law. At the Annual Conference of the National Conference of Commissioners on Uniform State Laws, a Uniform Act on Blood Tests to Determine Paternity was drafted and approved at a meeting held in San Francisco, 8—13 September 1952. This was approved by the American Bar Association at its meeting in San Francisco, 19 September 1952. The proposed Uniform Act has many commendable features. However, the following simplified version would be an improvement, in the opinion of this committee.

Uniform Act on Blood Tests in Cases of Disputed Parentage in Man

Be it enacted (use the proper enacting clause for the state).

SECTION 1. *Authority for Test.* In a civil action in which parentage is a relevant fact, the court, upon its own initiative or upon motion of any party in the action shall order the mother, child, and alleged father, to submit to blood tests.

SECTION 2. *Selection of Experts.* The tests shall be made by qualified experts in the field of blood grouping who shall be appointed by the court. For a list of such experts the court shall apply to the local medical association or to the American Medical Association. The expert may be called by the court to testify on the findings and shall be subject to cross-examination. Any party may demand that other qualified experts perform independent tests under order of the court.

SECTION 3. *Compensation of Expert Witnesses.* The compensation of each expert appointed by the court shall be fixed at a reasonable amount, and paid as the court shall order.

SECTION 4. *Effect of Test Results.* If the blood tests show that the alleged parent is not the parent of the child, such a finding shall be admissible as evidence. If the blood tests show the possibility of the alleged parent's parentage, the admission of such evidence shall be within the discretion of the court.

SECTION 5. *Effect on Presumption of Legitimacy.* The presumption of the legitimacy of a child born during wedlock is overcome if the court finds that the blood tests show that the husband is not the father of the child.

SECTION 6. *Applicability to Criminal Actions.* This act shall also be applicable in criminal cases in which the problem of parentage arises, such as cases of rape and kidnapping.

SECTION 7. *Severability Clause.* If any part of this act is declared invalid the remaining portion shall continue in full force and effect and shall be construed as being the entire act.

SECTION 8. *Short Title.* This act may be cited as the Uniform Act for Blood Tests in Disputed Parentage in Man.

SECTION 9. *Repeal.* All acts or parts of acts which are inconsistent with the provisions of this act are hereby repealed.

SECTION 10. *Time of Taking Effect.* This act shall take effect . . .

Summary and Conclusions

Because the use of two systems of nomenclature for the Rh-Hr system leads to confusion and misunderstanding, the Committee on Medicolegal Problems of the American Medical Association recommends the adoption of a single uniform system for medicolegal reports. It is recommended that unless and until some other convention can be agreed upon, the original Rh-Hr notations be retained as the standard and sole nomenclature for the preparation of approved medicolegal reports on Rh types.

It is recommended that routine medicolegal applications of blood grouping be restricted at present to tests for the four blood groups O, A, B and AB of the A-B-O system; the three types M, N, and MN of the M-N-S system; and factors **Rh_0**, **rh′**, **rh″**, and **hr′** of the Rh system. Tests for additional factors and systems are considered insufficiently established for routine use at present, although they may provide evidence in particular selected cases. Such evidence should be submitted in a separate letter not part of the standard report and should be presented with proper caution. Similarly, the value of rare combinations of groups providing indication of paternity, in contrast to the conventional use of the tests to establish only non-paternity, should be recognized in unusual cases but should be treated with caution in a separate communication not part of the definitive report.

References

1. Davidsohn, I., Levine, P., and Wiener, A. S.: *Medicolegal Application of Blood Grouping Tests, Bureau of Legal Medicine and Legislation, Committee on Medicolegal Problems*, J.A.M.A. **149**: 699-706 (June 14) 1952.
2. Wiener, A. S. and Wexler, I. B.: *Mosaic Structure of Red Blood Cell Agglutinogens*, Bact. Rev. **16**: 69-87 (June) 1952. Wiener, A. S.: *An Rh-Hr Syllabus*, New York, Grune & Stratton, Inc., 1954. Stormont, C.: *Linked Genes, Pseudoalleles and Blood Groups*, Am. Naturalist **89**: 105, 1955. Horowitz, N. H. and Owen, R. D.: *Physiological Aspects of Genetics*, Annual Rev. Physiol. **16**: 81-102, 1954. Irwin, M. R.: *Immunogenetics*, in *Biological Specificity and Growth*, edited by Elmer G. Butler, Princeton University Press, N.J., 1955.
3. Wiener, A. S.: *The Rh-Hr Blood Types: A Budget of Paradoxes*, J. Forensic Med. **2**: 224-242 (Oct.-Dec.) 1955. *The Terminology of the Rh Blood Group System*, editorial, *ibid.* **2**: 193-195 (Oct.-Dec.) 1955.
4. Wiener, A. S.: *Heredity and Nomenclature of the Rh-Hr Blood Types*, Bull. World Health Organ. **3**: 265-278, 1950.
5. Boyd, W. C.: *Compact Tabular Presentation of Mother-Child-Alleged Father Combinations which Establish Non-Paternity in the Rh Blood Group System*, Vox Sanguinis **5**: 99 (Aug.) 1955.
6. Levine, P., and others: *Gene Interaction Resulting in Suppression of Blood Group Substance B*, Blood **10**: 1100-1108 (Nov.) 1955.
7. Ottensooser, F. and Silberschmidt, G.: *Hemoglutinina Anti-N de Origem Vegetal*, Arq. biol. **38**: 13-14 (Jan.-Feb.) 1954.
8. Stormont, C.: *Personal communication to the Committee.*

The Electroencephalogram and Criminal Responsibility

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The electroencephalogram (EEG) may be introduced as evidence into court proceedings for one of two purposes. Most often it is introduced in accident cases as evidence of injury to the brain, but it may also be used in attempts to establish irresponsibility in criminal cases. This paper is concerned with the latter application; the former has been discussed in some detail elsewhere.[5, 6]

The criterion of criminal responsibility in courts in the United States and throughout the British Commonwealth is the M'Naghten formula[2].* To establish irresponsibility under the M'Naghten formula, the accused must be shown to have been laboring under such a defect of reason from disease of the mind as not to know the nature and quality of his act, or if he did know it, that he did not know that he was doing what was wrong. Practically, the first step in establishing a plea of irresponsibility is to show that the defendant was, at the time of his act, suffering from functional or organic psychosis (or more rarely, psychoneurosis or psychopathy) or from a defect of consciousness. It is at this point that the evidence of the EEG may be introduced, and it is the purpose of this paper to set forth the uses and limitations of the EEG in the development of such evidence. The discussion will deal with the usefulness of the EEG in establishing a diagnosis of:

(a) Functional mental disease (i.e. one not attributable to organic brain disease);
(b) Organic mental disease (attributable to brain disease); and
(c) Other disorders, especially epilepsy, which involve defects of consciousness.

Preliminarily however, some basic information about the EEG must be

* Some courts, however, have departed from the M'Naghten rule. In *Durham* v. *United States*, 214 F. 2d 862 (1954), the U.S. Court of Appeals for the District of Columbia adopted a broader test in what has become known as the 'Durham rule'. A jury must simply decide (a) whether an accused was suffering from 'a diseased or defective mental condition' when he committed the crime, and (b) if so, whether the crime was the 'product' of such abnormality. New Hampshire has followed a similar test since 1870.

set forth. The normal adult EEG,† recorded under standard conditions of relaxed wakefulness, shows two sorts of brain electrical activity:

(a) A more or less prominent and persistent rhythm at 8-13 cps (cycles per second) with a mean voltage of 35–50 microvolts, most prominent over the posterior half of the head, called the alpha rhythm, and

(b) Low-voltage fast activity (18–30 cps and less than 20 microvolts) most prominent in the precentral region, called beta activity (Appendix, Fig. 1A). The pattern changes markedly during drowsiness and sleep (Appendix, Fig. 1B).[1, 7, 9]

It must be emphasized that a normal EEG does not rule out brain disorder. EEGs are more often than not normal in many brain disorders (multiple sclerosis, arteriosclerosis, old brain lesions, etc.) and are even so in some epileptics between seizures. There would therefore be little point in introducing a normal EEG as evidence of brain normality.

EEG abnormalities may be divided, at the risk perhaps of oversimplification, into 3 general categories:

(a) Waves slower than 8 per second (Appendix, Fig. 3);
(b) Paroxysmal phenomena (Appendix, Fig. 4); and
(c) Unusually prominent fast activity (Appendix, Fig. 2).

Such abnormalities are further classified as diffuse or generalized (i.e. appearing over all areas of the brain) and focal (appearing in a limited area). Diffuse abnormalities are most often associated with generalized brain disease and idiopathic epilepsy, focal abnormalities with localized brain damage (traumatic injury, vascular accidents, and tumors) and symptomatic epilepsy. See Mundy-Castle[12] for a more thorough discussion of EEG abnormalities.

There is not however, universal agreement about what is abnormal. All focal (Appendix, Figs. 3C, 3D, and 4D) and paroxysmal (Appendix, Fig. 4) phenomena are universally considered abnormal; marked diffuse abnormality likewise. On the other hand, mild diffuse slow activity (Appendix, Fig. 3A) is not considered abnormal by some authorities.[18] Some workers consider diffuse fast activity (Appendix, Fig. 2) to be abnormal before age 40,[7] while others usually classify it as normal.[1, 18] It is generally considered to be normal after age 40, and it is frequently produced by such commonly-consumed drugs as the barbiturates. The incidence of mild diffuse slow activity among normal adults is about 8%, and that of diffuse fast activity about 6%.[8]

Disagreements among authorities make mildly irregular EEGs of dubious value in establishing pathology in a court of law. On the other hand, marked diffuse slow abnormality and all paroxysmal and focal abnormalities are of considerable value in establishing dysfunction.

† The EEGs of children, which cannot be judged by the same criteria as those of adults, will not be dealt with in this paper. For more extensive descriptions and illustrations of normal and abnormal EEGs of both adults and children see current textbooks and atlases.[1, 7, 9, 15, 16, 18] Mundy-Castle[12] has published an excellent, concise review in a recent issue of this Journal.

EEG and Functional Mental Disorders

The EEG cannot be used in the diagnosis of functional mental disease. The writer has reviewed the literature pertaining to this question elsewhere,[3] and will present only a brief summary here.

Rates of EEG abnormality found in studies of schizophrenics are generally higher than rates reported for normals (roughly 25—35% and 5—20%, respectively). Reports are variable, with rates for schizophrenics as low as 9% and as high as 60% reported. Data on other groups of functional psychotics (manic-depressives and involutionals) are insufficient to permit generalization. The majority of all EEG abnormalities among functional psychotics are of those non-specific types (Appendix, Figs. 2 and 3A) which are found most often among controls and about the significance of which there has been dispute. It is clear, therefore, that an abnormal EEG cannot be used as evidence of the existence of a functional psychosis.

The situation is little different with respect to the psychoneuroses and psychomatic disorders, where the majority of studies (7 of 11) have shown EEG abnormality rates no higher than those found in control groups.

Among psychopaths the situation is not quite the same. The findings have been more consistent and conclusive: 12 of 14 studies have shown EEG abnormality rates clustering in the range of 47—58%, and in the 5 instances when statistical comparison of psychopath and control groups has been possible, the differences have been found to be highly significant. EEGs of psychopathic criminals show the same incidence and kinds of abnormalities as those of non-criminal psychopaths; those of non-psychopathic criminals as a group do not differ significantly from those of controls. But here again the results are of little or no use for legal purposes, because the vast majority of the EEG abnormalities observed are of the non-specific, slightly slow type, and further, defects of reason are rarely of sufficient magnitude in non-psychotic mental disorders to meet the requirements of the M'Naghten formula. Mundy-Castle[13] has discussed these findings and their implications at greater length.

EEG and Organic Mental Disorders

A wide variety of organic brain disorders may give rise to, or precipitate, severe mental disorders. In any of them the EEG may reveal the disturbance of physiological function, but only in the diagnosis of some of them is it of regular value. These are brain tumors, and *acute* cerebral traumata (including the effects of brain operations such as lobotomy), cerebral vascular accidents, the encephalitides and meningitides and acute drug intoxication. (Epilepsy will be dealt with separately, below.) In these conditions the EEG is usually abnormal, and in the first three will often reveal the location of the disorder within the brain[1, 9, 16, 18] (Appendix, Figs. 3C and 3D). On the other hand, the EEG may return to normal

within a few days after the acute phase where the lesion or infectious condition has been mild, and the EEG is usually normal or only slightly abnormal even in the acute phase of some disorders which often give rise to mental syndromes, such as syphilis of the brain, cerebral arteriosclerosis, drug addiction (including chronic alcoholism) and old (chronic) brain damage unless seizures are associated with the primary disorder.

When an attempt is being made to establish the existence of organic brain disease, a grossly abnormal EEG provides strong support. However, the EEG is of no greater value here in establishing mental disorder *per se* than it is in the case of functional diseases.

EEG and Disorders of Consciousness: I. Epilepsy

It is hardly necessary any longer to point out that the EEG is the most reliable and valid instrument, next to a thorough clinical history, in the diagnosis of epilepsy. Since the essential feature of epileptic seizures is the abnormal discharge of electrical activity in the brain, the EEG is ideally suited for this purpose. Paroxysmal phenomena (Appendix, Fig. 4) are the signs par excellence of epileptogenic processes. EEG technique does however, have limitations: (a) seizure discharges arising in deep structures of the brain, and therefore inaccessible to the pick-up electrodes on the surface of the cranium, usually cannot be located precisely and are sometimes missed completely, and (b) since epilepsy is by nature a paroxysmal disorder, there are times in most patients when no seizure discharges occur, and EEGs recorded at those times will not reveal abnormality. Repeated recordings and various procedures devised to activate latent abnormality[7, 9, 15, 16] have succeeded in reducing errors of the second type, but some failures must still be expected.

The most useful application of the EEG in dealing with a plea of irresponsibility would be in assisting to establish a diagnosis where it is claimed that the accused committed his criminal act while unconscious during an epileptic seizure and could therefore not possibly have known the nature and quality of his act. In such cases the nature of the seizures to which the accused is subject are important. Criminal acts cannot be committed during simple or myoclonic *petit mal* attacks or during the tonic-clonic phase of a *grand mal* seizure. It is conceivable that destructive acts might be committed during the period of confusion which follows some *grand mal* seizures, but the victim's actions in such confusional states are usually more or less disorganized and ineffectual. Focal seizures arising in the frontal, parietal or occipital sectors of the brain are likewise rarely associated with destructive behaviour. Ictal violence and destructiveness most often occur during those seizures designated 'psychomotor'. The vast majority of psychomotor seizures are of temporal lobe origin (Appendix, Fig. 4D), as has been demonstrated principally by electroencephalography.

There is not space to describe the great variety of symptoms which may occur during temporal-lobe seizures,[7, 14, 15] but it is only with acts of violence that we are concerned here. The nature of such seizures can best be illustrated with an example, quoted from Gastaut *et al.*[4]

Case No. 7: M. X., a young man of 19 years, confined for repeated violent attacks on various persons, the last of which just fell short of ending in homicide, the patient being armed. At EEG examination a right temporal focus of slow waves and sporadic slow spikes was observed.

During Cardiazol (Metrazol) activation the subject suddenly showed an 'investigative reaction' during which he looked to the right and to the left with an uneasy air, then became frightened and finally terror-stricken. During this time he cried out angry words . . . and at the same time rushed forward, fighting furiously with the attendants who tried to control him. There was total amnesia for the seizure.

Such cases are not common, constituting only about 2—3% of all persons exhibiting psychomotor seizures, who in turn constitute about 25% of all epileptics.[7] Since the incidence of epilepsy in the general population is 0.5%, only 3 or 4 persons in 100,000 are likely to commit such unconscious acts of violence, and only a few such acts will result in the commission of prosecutable crimes. However, such cases are likely to be dramatic. The EEG and other laboratory procedures, such as described with the case above, together with a complete clinical history, should suffice in most cases to establish the ictal nature of the act, but an accurate description of the accused's behaviour during the commission of the act would be of great help, if not essential with some juries.

The incidence of behaviour disorders in psychomotor epileptics during interseizure periods is high (45—50%).[7, 13] Gastaut *et al.*[4] observed that a common trait is the display of violent and uncontrolled outbursts of rage, which occurred in 50% of their carefully studied series of 60 patients. One of their cases was described as follows:

Case No. 1: L. P., age 36. Clinical vertiginous seizures followed by confusional state with automatisms, since age 26. Sometimes secondary generalized seizures. Frequency of minor not-generalized seizures: about 1 a week. *Etiology*: cranial trauma at age 23. *Neurological*: right hemiparesis. *Pneumoencephalogram*: deviation of the ventricular system to the right. *EEG*: background rhythms normal, no focal activity. Cardiazol (Metrazol) provoked delta waves and sporadic slow spikes in the left temporal area. Then a discharge of slow waves appeared, rapidly spreading to involve the entire left hemisphere and accompanied by a clinical psychomotor seizure.

Most of the time the patient was listless. He would remain depressed for several weeks and then suddenly, without apparent reason, go into a terrible rage and break everything that came to hand. The patient himself says: 'At those times I am capable of killing.' He cannot explain his impulses, and regrets them later.

Such cases as this present a somewhat more difficult medico-legal problem. Gastaut is convinced that such outbursts of rage and violence are not ictal phenomena on the grounds that (a) the pateint is not amnesic for the episode, (b) they cannot be induced by Metrazol injection, and (c) in a few

cases EEGs have been recorded during such episodes without showing seizure discharges. But is the patient responsible? Can he control his actions? The handling of such cases would undoubtedly vary in different jurisdictions, the accused perhaps being held irresponsible where irresistible impulse is an acceptable defence.

In addition to temporal-lobe psychomoror epilepsy, Gibbs and Gibbs have described a constellation of paroxysmal disorders which they group under the term 'thalamic or hypothalamic epilepsy', and which they associate with a peculiar EEG phenomenon, 14 and 6 per second positive spikes. These spikes, which are usually seen only during sleep, arise in the diencephalon, they believe. Their data show that 10.5% of 427 patients with 14 and 6 per second spikes were subject to paroxysmal rage reactions with attack behaviour of an organized and well-directed sort, for which the patient was afterwards partially or totally amnesic. Of such patients, 4 were institutionalized for murder and 12 others because they were considered dangerous. Schwade and Geiger[17] have described a case of matricide by a 14-year-old boy who displayed almost continuous 14 and 6 per second spikes both while awake and asleep. This category of thalamic or hypothalamic epilepsy with 14 and 6 per second spikes is not as widely recognized by epileptologists as is temporal-lobe psychomotor epilepsy.

EEG and Disorders of Consciousness: II.

Not all paroxysmal defects of consciousness are of epileptic nature.* Syncopal attacks are not uncommon. Periodic 'blackouts' due to hypoglycemia are rarer, but not unknown. During such periods of unconsciousness or obnubilation, effectual muscular behaviour is rare, but well-organized actions, for which the victim is later amnesic, can occur. Hill and Sargant[10] have presented a case of matricide occurring under such conditions, which well illustrates the role of laboratory procedure in demonstrating the existence of a true cerebral dysfunction under the conditions under which the crime was committed. A brief account of their case will be found in Mundy-Castle's paper.[13]

Summary

The usefulness of the EEG as an adjunctive instrument in establishing diagnoses of functional and organic mental disease and epilepsy, or other disorders associated with defects of consciousness, has been discussed. The following conclusions are drawn:

1. The EEG is of no value at the present time in the diagnosis or evaluation of functional mental disease.

* Lennox reviewed the subject of amnesia in relation to medico-legal problems some years ago.[11]

2. Abnormal EEGs, especially those showing marked slow activity or focal disturbances of brain electrical function, provide supportive evidence of the existence, and sometimes of the location, of organic disorder of the brain. The exact nature of the disorder cannot be determined from the EEG, nor can the coexistence of mental disorder be inferred.

3. The EEG is of greatest value in the diagnosis of epileptic disorders. Especially when activation procedures are used, the exact nature of patients' seizures may be determined. The physiological conditions under which a crime has been committed may be reproduced in the laboratory, and may provide strong evidence to support a plea of irresponsibility.

4. A normal EEG cannot be considered conclusive evidence of the absence of brain disease.

References

1. Cohn, R. (1949): *Clinical Electroencephalography*, New York: McGraw-Hill.
2. Davidson, H. A. (1952): *Forensic Psychiatry*, New York: Ronald.
3. Ellingson, R. J. (1954): *The Incidence of EEG Abnormality among Patients with Mental Disorders of Apparently Nonorganic Origin: A Critical Review*, Amer. J. Psychiat., **111,** 263.
4. Gastaut, H. Morin, G. and Lesèvre, N. (1955): *Étude du Comportement des Épileptiques Psycho-moteurs dans l'Intervalle de leurs Crises*, An. Méd.-Psychol., **113,** 1.
5. Gibbs, F. A. (1946): *Medicolegal Aspects of Electroencephalography*, Canad. Bar Rev., **24,** 359.
6. Gibbs, F. A. (1946): *Medicolegal Aspects of Electroencephalography*, J. Clin Psychopathol., **8,** 58.
7. Gibbs, F. A. and Gibbs, E. L. (1950, 1952): *Atlas of Electroencephalography*, 2nd Ed., Cambridge, Mass.: Addison-Wesley, 2 vols.
8. Gibbs, F. A., Gibbs, E. L. and Lennox, W. G. (1943): *Electroencephalographic Classification of Epileptic Patients and Control Subjects*, Arch. Neurol. Psychiat., **50,** 111.
9. Hill, D. and Parr, G., Eds. (1950): *Electroencephalography*. London: Macdonald.
10. Hill, D. and Sargant, W. (1943): *A Case of Matricide*, Lancet, **244,** 526.
11. Lennox, W. G. (1943): *Amnesia, Real and Feigned*, Amer. J. Psychiat., **99,** 732.
12. Mundy-Castle, A. C. (1954): J. Forensic Med., **1,** 338.
13. Mundy-Castle, A. C. (1955): J. Forensic Med., **2,** 95.
14. Penfield, W. and Kristiansen, K. (1951): *Epileptic Seizure Patterns*. Springfield, Ill.: Charles C. Thomas.
15. Penfield, W. and Jasper, H. H. (1954): *Epilepsy and the Functional Anatomy of the Human Brain*. Boston: Little, Brown.
16. Schwab, R. S. (1951): *Electroencephalography in Clinical Practice*. Philadelphia: Saunders.
17. Schwade, E. D. and Geiger, S. G. (1953): *Matricide with Electroencephalographic Evidence of Thalamic or Hypothalamic Disorder*, Dis. Nerv. Syst., **14,** 18.
18. Strauss, H., Ostow, M. and Greenstein, L. (1952): *Diagnostic Electroencephalography*. New York: Grune & Stratton.

(*For* Appendix *see pp. 122-130.*)

Appendix

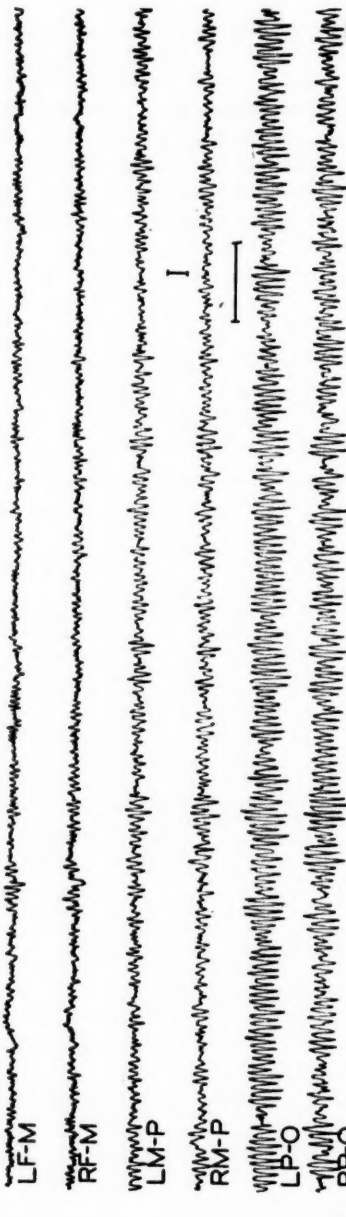


Fig. 1. Normal adult EEGs.
(A) Female, age 57, awake. Note strong, steady alpha rhythm, of maximal voltage posteriorly. Beta activity can be seen anteriorly, in the upper 2 tracings.

Fig. 1. Normal adult EEGs.
(B) : Male, age 31, light sleep. So-called 'spindle bursts' of 14 per second waves are most clearly seen in the motor-parietal region. This distribution is typical. In deeper sleep 'spindle bursts' become less prominent and slow waves (1—3 per second) dominate the record.
Labelling of tracings in this and the following figures is according to the following code:
L=left, R=right, F=frontal, M=motor (precentral), C=central, P=parietal, O=occipital, T=temporal, AT=anterior-temporal, PT=posterior-temporal. In 'unipolar' recording, where the second electrode of each pair is a 'neutral' reference electrode (conjoined ear-lobe leads in these illustrations), only the 'active' electrode is indicated, i.e. in Figs. 3C, 4C, and 4E.
The horizontal calibration lines indicate 1 second and the vertical indicate 50μV in all figures.

Fig. 2. Diffuse fast activity. Male, age 28, awake, no sedation. Idiopathic epilepsy, *grand mal* (1 seizure a year). This degree of fast activity is distinctly beyond normal limits for the patient's age.

LF-AT
RF-AT
LAT-PT
RAT-PT
LPT-O
RPT-O
1
1
1

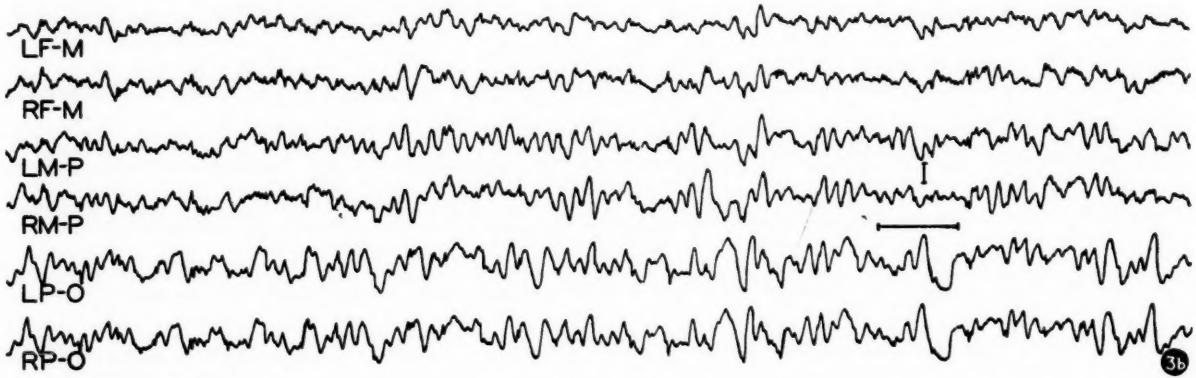


Fig. 3. Slow wave abnormalities.
(B) ***Marked diffuse slow activity, maximal posteriorly.*** Male age 40, awake. Acute virus encephalitis. This degree of abnormality is almost never seen in normal adults.

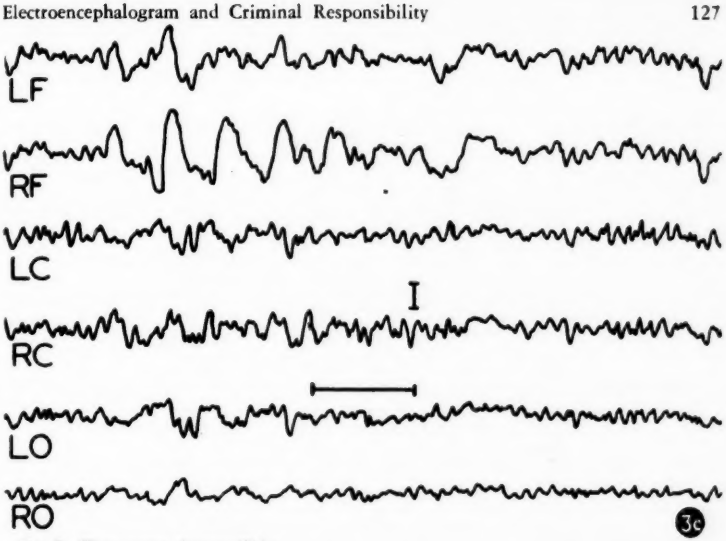
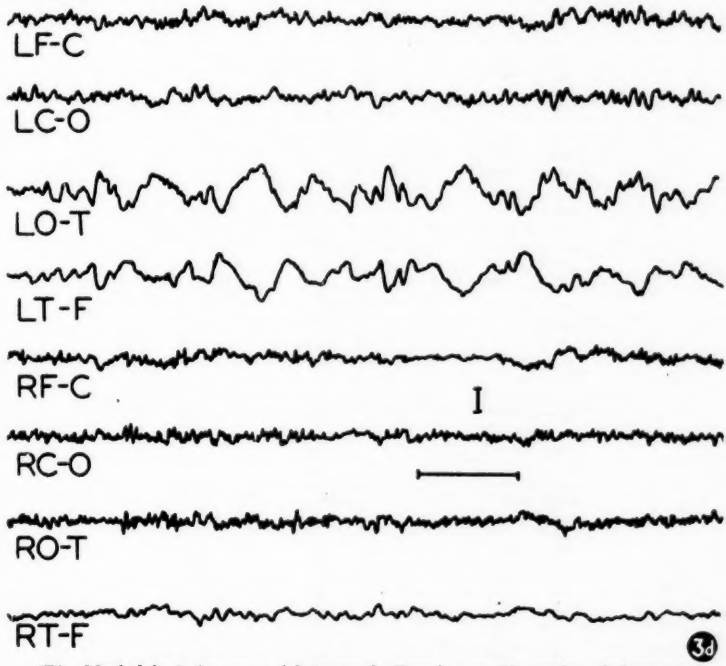


Fig. 3. Slow wave abnormalities.

(C) *Marked focal slow waves, right frontal, with disturbance of function in adjacent areas.* Male, age 33, awake. Right frontal glioblastoma multiforme with Jacksonian seizures involving the left face and arm. This illustrates localization by the unipolar method: the lesion is in the area where the slow waves are of highest voltage and longest duration.



(D) *Marked focal slow waves, left temporal.* Female, age 38, awake. Subacute subarachnoid hemorrhage; right hemiplegia and aphasia. This illustrates localization by the bipolar method: the lesion is in the area of the electrode common to the 2 tracings in which the slow waves appear, and because of the instrumental arrangement the slow waves appear out-of-phase in those 2 tracings.

Fig. 4. *'Seizure discharges.'*
(A) *Diffuse* 3 *per second spike-and-wave complexes: clinical* petit mal *seizure of* 17 *seconds duration.* Female, age 10. Idiopathic epilepsy, *grand mal* and *petit mal.* The 'classical' spike-and-wave pattern shown is much more commonly seen in children than adults. Note that in this bipolar recording the spike-and-wave complexes are inverted in the first 2 channels. Note also that some of the spikes are multiple, which is more common in patients showing *grand mal* seizures than in those with only *petit mal* seizures.

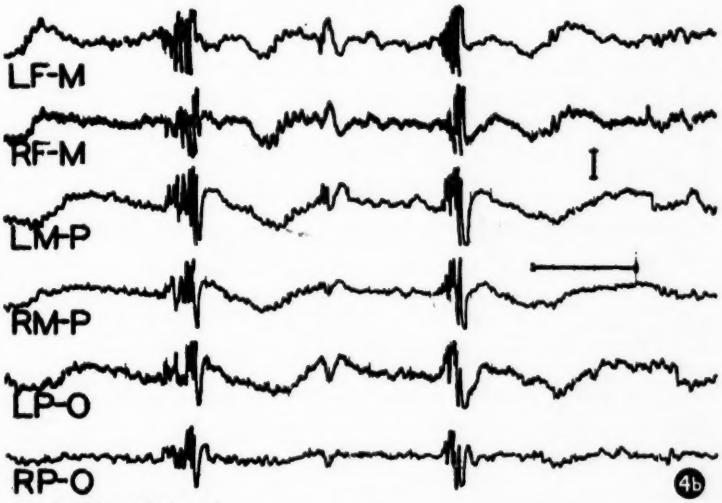
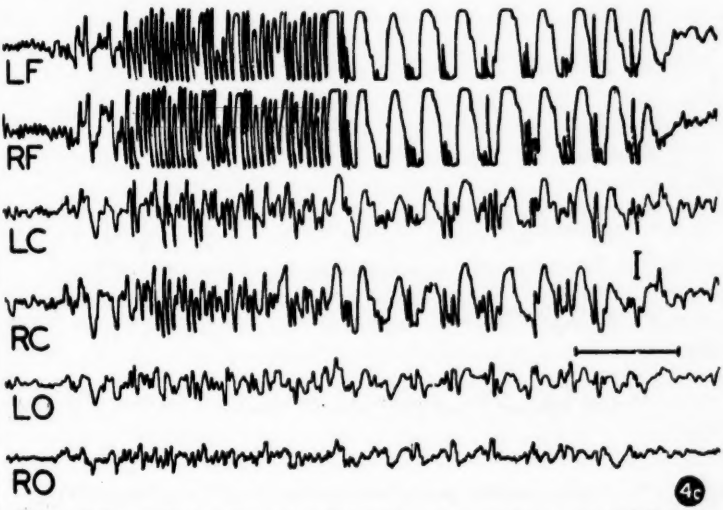


Fig. 4. *'Seizure discharges.'*
(B) *Diffuse multiple spikes.* Male, age 33, asleep. Idiopathic epilepsy, *grand mal.* These spike bursts resemble muscle potential, but in the original record are clearly not, having a frequency of 30 per second.



(C) *Diffuse multiple spikes and multiple-spike-and-wave complexes, maximal frontally.* Male, age 41, awake. Idiopathic epilepsy, *grand mal.* Both this and the preceding phenomenon are relatively rare. Normal interseizure EEGs are more common in idiopathic *grand mal* than in any other type of epilepsy.

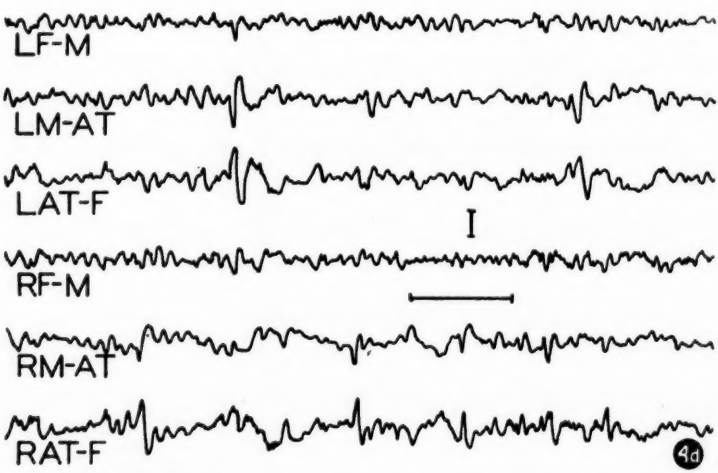
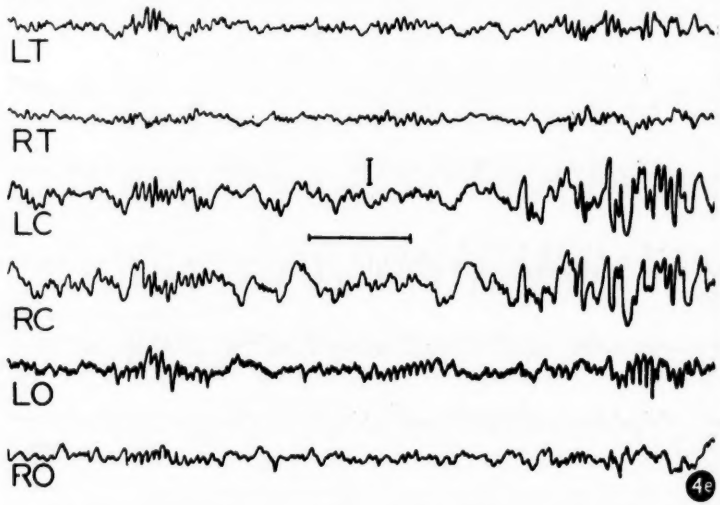


Fig. 4. '*Seizure discharges.*'

(D) *Focal spikes, alternating bitemporal.* Male, age 41, asleep. Sensory-psychomotor seizures, consisting of an olfactory hallucination followed by a period of amnesia lasting for several minutes, during which the patient often continues to display effectual behavior, such as driving. This record illustrates localization by the bipolar 'triangulation' method. Note that the spikes in the right temporal area are of different amplitude, duration, and occur at different times from those on the left. The spikes in this record are of the type often called 'slow spikes' or 'sharp waves' (compare with the spikes in 4 B).



(E) 14 *per second positive spikes.* Male, age 10, asleep. 'Nervousness' and dizzy spells since age 6; no periods of unconsciousness or convulsive phenomena have been reported. Note the strong sleep spindle activity in the LC and RC tracings. The 14 per second spikes are especially prominent in the LO tracing.

Poisoning by 'Alcohol'

Particularly Methyl Alcohol

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The term 'alcohol' is normally loosely used to mean ethyl alcohol and certainly has this interpretation so far as the general public is concerned. Ethyl alcohol is, however, only one of a series of compounds of increasing molecular weight with similar chemical properties and designated chemically as 'alcohols'. The lower members of the series have hypnotic properties and act as depressants of the central nervous system. The toxic properties increase, as a rule, with increasing molecular weight up to a certain limit, after which there is a decrease. However, an exception must be made in the case of methyl alcohol which is somewhat more toxic than the more commonly encountered ethyl alcohol which forms the basis of all alcoholic beverages. Poisoning by ethyl alcohol is by no means uncommon although death as a direct result is somewhat rare. Methyl alcohol, however, differs markedly from ethyl alcohol in its effects and is much more dangerous. According to Cushny [1] this arises from its slower oxidation in the system and consequently its more prolonged action. As a result cases of death from methyl alcohol are by no means uncommon, and many cases of 'mass poisoning' by methyl alcohol have been recorded.[3]

Methyl alcohol may be obtained from the 'pyroligneous liquor' collected during the destructive distillation of wood, but it may also be manufactured synthetically from carbon dioxide and/or carbon monoxide and hydrogen. It finds a wide use in industry as a general solvent, especially for resins and varnishes, and as a raw material for the manufacture of organic compounds such as formaldehyde, formic acid, drugs and dyes. Because methyl alcohol can be obtained from such sources it is sometimes drunk by those who are unable to get ordinary alcohol or by those who are misled by the term 'alcohol' and do not appreciate the fatal dangers involved in drinking methyl alcohol.

In this paper it is not intended to deal specifically with the effects of drinking ethyl alcohol, as such, nor to discuss the problem of ethyl alcohol in blood as an index of intoxication. During the period 1948-1952 there

were 5 cases involving methyl alcohol poisoning and 1 case of poisoning by butyl alcohol, examined in the Government Chemical Laboratories, Johannesburg. There was one case in which 'methylated' spirits caused the death of a child. It must be pointed out that, in South Africa, 'methylated' spirits is very rarely prepared with methyl alcohol (even in the crude form of 'wood naphtha') and is usually manufactured with pyridine and/or butyl alcohol. There was one case (No. 2109/49) in which the stomach contents of a deceased African male was found to contain 1093 p.p.m. (mg. per kg.) of ethyl alcohol, but the history of the case showed that he actually died from stab wounds received in a drunken brawl.

Method of Determination

It is necessary to describe the method used from the determination since the general procedure adopted has an important bearing upon the whole problem of alcoholic intoxication.

In cases of death from suspected poisoning the liver, the kidneys, the stomach and frequently the intestines are submitted to the Laboratory for examination, together with any information available concerning the case. The organs are weighed separately and some preliminary tests carried out in an attempt to obtain some indication of the possible poison. The various organs are then finely divided and aliquot weights taken for the determinations required. In the case of alcohols, these samples are separately steam distilled. The distillate is then acidified and re-distilled to yield about 100 ml. distillate. In this way volatile bases are removed and this second distillate will then contain any alcohols originally present together with any volatile acids, aldehydes and ketones. These last impurities may be removed by treating the second distillate with Nessler's reagent in excess. After standing for some hours, the solution is re-distilled to a volume of about 60 ml. This distillate is again acidified and distilled to a volume of 20 or 25 ml. This final distillate will now contain all the alcohol originally present in the organ examined and will be free from interfering substances. This procedure is essentially the same as that recommended by a 'Panel of Analysts' in Britain for the determination of alcohol in blood and urine.[2]

The total amount of alcohol is determined by the usual bichromate method, using an aliquot of the final distillate. The residual distillate is concentrated to 5 ml. by distillation and both the specific gravity and refractive index determined. From these figures it is possible to ascertain whether methyl or ethyl alcohol is present and if both are present to calculate the proportions of each. The presence of methyl alcohol can be confirmed by oxidizing the methyl alcohol to formaldehyde and applying suitable tests to detect its presence.

Methyl Alcohol Poisoning

The symptoms of methyl alcohol poisoning last much longer than those due to ethyl alcohol and are probably due to the slower oxidation of methyl alcohol so that the resulting products are retained in the system for a longer period. In some cases the symptoms appear suddenly in a severe form, the patient becoming rapidly unconscious and death supervening rapidly. However, the most frequent and important symptom of

methyl alcohol poisoning is blindness and this may occur after one dose or after a series of small, non-toxic doses.

Individuals differ in their reactions to the ingestion of methyl alcohol, some showing an idiosyncrasy and others a tolerance. The fatal dose cannot, therefore, be stated with any degree of certainty but it appears to be between 30 and 100 gm. This is a problem which is ever present in cases of death by poisoning and seems incapable of a decisive answer. In fatal cases of methyl alcohol poisoning, death may occur within half an hour, but it usually takes place between 24 and 36 hours.

Alcohols of higher molecular weight than ethyl alcohol are not usually met with in poisoning cases; but during 1948-1952 there was one case which involved poisoning from butyl alcohol. This alcohol is much more toxic than ethyl alcohol and shows the same effects in a much more marked form.

Death from Alcoholic Poisoning

Since methyl alcohol is more slowly oxidized in the system than ethyl alcohol, it is possible to detect the former alcohol in the organs a few days after ingestion. The results obtained in the viscera are tabulated in Table I in terms of parts per million (mg. per kg.).

Table I: Alcoholic Poisoning

Case	*Race and Sex*	*Alcohol*	*Liver (L)*	*Kidneys (K)*	*Stomach (S)*	*Intestines (I)*
3716/51	N.M.	Butyl	3600	3600	4600	5100
4297/52	N.M. CL.(*a*)	Ethyl	—	—	—	12550(*b*)
4792/52	N.F.	Methyl	3681	—	—	2816
4893/52	N.M.	Methyl	4448	3959	3959	3880
4894/52	N.M.	Methyl	2200	2250	2250	1800
		Ethyl	700	700	700	500
4895/52	N.M.	Methyl	690	500	500	600
		Ethyl	150	100	100	100
5840/52	N.M.	Methyl	648	620	620	620

N. = Native (i.e. African)
M. = Male.
F. = Female.
CL. = Child.

(*a*) Blood = 7668 p.p.m. ethyl alcohol.
(*b*) Ethyl alcohol in colon.

In the first place it will be noted that all the cases involve Africans and it must be concluded that little discrimination has been exercised by the victims in regard to a liquid which bears the generic name of 'alcohol' and which can produce the symptoms of intoxication. It is apparent that the dangers attached to such actions cannot be fully realized. Secondly the results indicate that the alcohols as a general rule become uniformly distributed through the system. The average values of the ratios L:K, L:S and L:I were found to be 0.99, 1.09 and 1.18 respectively, and these do not differ significantly from unity. This uniform distribution is more pronounced than has been encountered with other poisons. In those cases

where both methyl and ethyl alcohol were present, the concentrations of methyl alcohol were higher than those of ethyl alcohol. This would appear to support the contention that ethyl alcohol is more rapidly eliminated from the system than methyl alcohol but in the above cases it must be remembered that no information is available concerning the proportions of the alcohols actually ingested.

Case No. 3716/51 is of interest as an example of death involving butyl alcohol poisoning. The deceased was last seen alive 'under the influence of liquor' and at the post-mortem examination 'a strong smell of ? chloroform ? alcohol was noted in the stomach'. An examination of a bottle of liquid, found on the deceased and initially suspected to contain cane spirit, revealed that it was actually a 'brake fluid', consisting of a mixture of butyl alcohol, castor oil and glycol. Castor oil and glycol were proved to be present in the stomach contents and there was no doubt that the deceased died from the effects of drinking 'brake fluid', of which the toxic constituent was the butyl alcohol. Butyl alcohol has a somewhat powerful and persistent odour.

Case No. 4297/52 involved the death of an African male child to whom an enema of methylated spirits had been administered. This accounts for the high ethyl alcohol content of the colon, viz. 12,550 p.p.m. The blood alcohol content was also extremely high, viz. 7,668 p.p.m. The fact that no methyl alcohol was detected is in conformity with rare occurrence of methylated spirits prepared with methyl alcohol as the denaturing agent. After the administration of the enema, the child 'soon became comatose and died'. It was reported that the bowel mucosa was congested.

Case 4792/52 was described as one of multiple cases of poisoning. Within a period of 4 days 3 other persons at the same address 'also passed away' and in each case there was vomiting and frothing at the mouth. The above deceased died within about 12 hours. There were no characteristic post-mortem findings but in view of the multiple deaths a chemical examination 'seemed desirable'. There was no doubt from the results that death was due to methyl alcohol poisoning.

The following 3 cases (Nos. 4983-5/52) were also an example of multiple poisoning, and the post-mortem examinations 'did not show any signs of poisoning'. In case No. 4983/52 methyl alcohol alone was found in the organs submitted for analysis. In the 2 remaining cases both methyl and ethyl alcohol were found to be present in the organs. No histories of these cases were supplied and it seems logical to assume, therefore, that the two latter victims had been drinking ethyl alcohol as well as methyl alcohol and from the amounts of about 700 p.p.m. (=0.07%) and 150 p.p.m. (=0.015%) of ethyl alcohol found in the organs, it seems logical to assume that the deaths were due to methyl alcohol poisoning. In passing it may be said that, in cases of intoxication, the blood content of ethyl alcohol ranges from about 0.15 % to 0.25%.

Case No. 5840/52 provides an illuminating example of methyl alcohol poisoning. The deceased worked in a laboratory where he had access to methyl alcohol, and took the opportunity of drinking it. He was taken suddenly ill and died within 'a few hours', but became 'completely blind' before death. There was congestion of the brains, lungs, kidneys and spleen.

Diagnosis of Methyl Alcohol Poisoning

Because methyl alcohol is more slowly oxidized than ethyl alcohol in the system, it is often possible to detect methyl alcohol in the tissues for a fews days after ingestion, but considerable difficulty is attached to a correct diagnosis from the symptoms alone. In some cases there is a 'latent' period before symptoms appear, while in other cases the symptoms appear suddenly, the patient passing rapidly into a coma with cyanosis and failing respiration. There may be depression, weakness, nausea and severe gastric pain. Webster[3] states that the most frequent and important single symptom of methyl alcohol poisoning is blindness, which occurs in a large percentage of cases.

In cases of poisoning a considerable amount of methyl alcohol is slowly oxidized to formic acid, which may be eliminated in the urine and this may be used for diagnostic purposes during life. However, a quantitative determination must be made since the urine normally contains a small amount of formic acid. Webster[3] quotes Autenreith to the effect that a person, on the average, eliminates normally about 0.25 grain of formic acid in the urine in 24 hours, but there is a marked increase following intake of methyl alcohol. There is also a condition of general acidosis.

At autopsy there is usually cyanosis of the head and face and rigor is well marked. The lungs are hyperaemic and oedematous and the liver may be enlarged and fatty. The mucous membranes of the gastro-intestinal tract may be slightly haemorrhagic and hyperaemia of the bladder mucosa appears to be a more or less constant feature of methyl alcohol poisoning.

In cases where the victim of poisoning is alive every effort must be made to eliminate this alcohol from the system, e.g. by stomach wash-outs. Diuresis must be stimulated and alkaline therapy must be adopted to combat the acidosis which develops.

References

1. Cushny, R. A. (1932): *Pharmacology and Therapeutics*, London: J. & A. Churchill Ltd.
2. Kent-Jones, D. W. and Taylor, G. (1954): *The Determination of Alcohol in Blood and Urine*, Anal., **79**, 121.
3. Webster, R. W. (1930): *Legal Medicine and Toxicology*, London: W. B. Saunders Company.

CORRESPONDENCE

Impartial Medical Testimony

To the Editor: As one who has previously advocated an independent neutral panel of expert psychiatric witnesses in Court cases (this Journal, **1**, 205) I have naturally been interested in the report of the New York Bar Association on *Impartial Medical Testimony*, on which you comment in a recent editorial.

While I am not qualified to speak except in the psychiatric field, I nevertheless regret that you find it necessary to raise what are, in effect, entirely theoretical objections to this most promising scheme. However, as in this respect, all experts are in the same position, viz. only qualified to speak for their particular branch, I feel justified in attempting to rebut the faint praise with which you damn the scheme.

Your objections boil down to:

1. Expense.
2. The panel will acquire too much prestige.
3. The best men may not get on to the panel.

Permit me to make some brief comments on these points.

1. The expense should be borne by the State. It will more than recoup itself by the time and money saved through the shortening of trials. In any case, the money spent will be a mere fraction of what is at present spent by litigants and accused persons on medical expert witness fees. This now forms an appreciable item in the cost of litigation, and its abolition will ease the litigant's financial burden appreciably.

2. It is of course necessary that the panel should have prestige and should speak with authority. But, however high its standing, I cannot visualize our lawyers ever being too overawed to cross-examine, especially as nothing prevents them from receiving advice from their own experts.

3. There can never be unanimity as to who are the best men, but it is reasonable to assume that those appointed will be a representative cross-section of the best men in their special field. Those omitted will be kept fully occupied in advising the legal profession how to refute the evidence of the panel.

Your appeal not to discard lightly 'sound and well-tried principles, tested over the centuries' would have more force if it was not notorious that our present system is crying out for reform, while your desire to see unethical conduct in the witness-box adequately punished, is, I am afraid, mere wishful thinking.

This letter reflects my personal opinion and is not written in my official capacity.

Umgeni Waterfall Institution,
Howick, Natal.
29 June 1956.

M. Minde,
Physician Superintendent.